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NEWS 10 Dec 15 2001 STN Pricing

NEWS 11 Dec 17 Merged CEABA-VTB for chemical engineering and biotechnology

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=> file reg

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SINCE FILE TOTAL ENTRY SESSION 1.05 1.05

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=> s polyethylene glycol

6215 POLYETHYLENE 38245 GLYCOL 715 GLYCOLS

38245 GLYCOL

(GLYCOL OR GLYCOLS)
5185 POLYETHYLENE GLYCOL
(POLYETHYLENE(W)GLYCOL)

L1

```
=> s polypropylene glycol
          2637 POLYPROPYLENE
         38245 GLYCOL
           715 GLYCOLS
         38245 GLYCOL
                  (GLYCOL OR GLYCOLS)
          2535 POLYPROPYLENE GLYCOL
L2
                 (POLYPROPYLENE (W) GLYCOL)
=> s polybutylene glycol
            81 POLYBUTYLENE
         38245 GLYCOL
           715 GLYCOLS
         38245 GLYCOL
                  (GLYCOL OR GLYCOLS)
L3
            64 POLYBUTYLENE GLYCOL
                 (POLYBUTYLENE (W) GLYCOL)
=> s polypentylene glycol
             2 POLYPENTYLENE
         38245 GLYCOL
           715 GLYCOLS
         38245 GLYCOL
                  (GLYCOL OR GLYCOLS)
             1 POLYPENTYLENE GLYCOL
L4
                  (POLYPENTYLENE (W) GLYCOL)
=> polyhexylene glycol
POLYHEXYLENE IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).
=> s polyhexylene glycol
             0 POLYHEXYLENE
         38245 GLYCOL
           715 GLYCOLS
         38245 GLYCOL
                 (GLYCOL OR GLYCOLS)
               POLYHEXYLENE GLYCOL
L5
                  (POLYHEXYLENE (W) GLYCOL)
=> s polyheptylene glycol
             0 POLYHEPTYLENE
         38245 GLYCOL
           715 GLYCOLS
         38245 GLYCOL
                 (GLYCOL OR GLYCOLS)
             O POLYHEPTYLENE GLYCOL
L6
                  (POLYHEPTYLENE (W) GLYCOL)
=> s polyoctylene glycol
```

0 POLYOCTYLENE

38245 GLYCOL 715 GLYCOLS 38245 GLYCOL

(GLYCOL OR GLYCOLS) O POLYOCTYLENE GLYCOL (POLYOCTYLENE (W) GLYCOL)

=> s polynonylene glycol

0 POLYNONYLENE

38245 GLYCOL 715 GLYCOLS 38245 GLYCOL

(GLYCOL OR GLYCOLS)

 $\Gamma8$

L7

O POLYNONYLENE GLYCOL (POLYNONYLENE (W) GLYCOL)

=> s polydecylene glycol

0 POLYDECYLENE

38245 GLYCOL 715 GLYCOLS 38245 GLYCOL

(GLYCOL OR GLYCOLS)

L9

O POLYDECYLENE GLYCOL (POLYDECYLENE (W) GLYCOL)

=> file bioscience

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COST IN U.S. DOLLARS

SINCE FILE TOTAL ENTRY SESSION 70.57

FULL ESTIMATED COST

71.62

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10 FILES SEARCHED...

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12 FILES SEARCHED...
  13 FILES SEARCHED...
  15 FILES SEARCHED...
  16 FILES SEARCHED...
  24 FILES SEARCHED...
  25 FILES SEARCHED...
  28 FILES SEARCHED...
  34 FILES SEARCHED...
  36 FILES SEARCHED...
  41 FILES SEARCHED...
  43 FILES SEARCHED...
  46 FILES SEARCHED...
  47 FILES SEARCHED...
  48 FILES SEARCHED...
  51 FILES SEARCHED...
  53 FILES SEARCHED...
        445916 L1 OR L2 OR L3 OR L4 OR PEG OR (POLYALKYLENE (W) GLYCOL?)
=> s ((spinal or spine?) or neuro? or nerve? or axon?) (w) (injury or
injuries or impairment? or impair? or damage?)
   8 FILES SEARCHED...
  12 FILES SEARCHED...
  20 FILES SEARCHED...
  28 FILES SEARCHED...
  31 FILES SEARCHED...
  37 FILES SEARCHED...
  41 FILES SEARCHED...
  45 FILES SEARCHED...
  50 FILES SEARCHED...
        129303 ((SPINAL OR SPINE?) OR NEURO? OR NERVE? OR AXON?) (W) (INJURY
               OR INJURIES OR IMPAIRMENT? OR IMPAIR? OR DAMAGE?)
=> s 110 and 111
  31 FILES SEARCHED...
          497 L10 AND L11
L12
=> d 1-5 kwic
     ANSWER 1 OF 497 ADISALERTS COPYRIGHT 2001 (ADIS)
L12
TX.
     . . definite or laboratory-supported definite multiple sclerosis. They
     had secondary progressive disease, with or without exacerbations,
     accompanied by gradual progression of neurologic
     impairment. Disease duration was > 1 year. Patients had a Kurtzke
     Expanded Disability Status Scale (EDSS) score of 3-7.5 (mean 5.2)..
TX
     Results:
     EDSS and Nine Hole Peg Test (9HPT)
     No significant between-group differences were observed in best or
averaged
     EDSS and 9HPT scores after treatment with AG 284.
     Gadolinium-enhanced.
L12 ANSWER 2 OF 497 BIOSIS COPYRIGHT 2001 BIOSIS
    . . and Levi, 1998). However, cyclosporin A (CsA), a potent inhibitor
AB.
of
     the permeability transition in liver mitochondria, only protects against
     neuronal injury by limited doses of glutamate and
     selected ischemic paradigms. The lack of consistent CsA inhibition of the
     mitochondrial permeability transition. . . prevent Ca2+-induced
```

depolarization or to repolarize mitochondria when mitochondria were depolarized excessively. Similarly, CsA failed to prevent mitochondrial swelling or PEG-induced shrinkage after swelling when the Ca2+ challenge produced a strong, sustained depolarization. Thus in brain mitochondria CsA may be effective.

- L12 ANSWER 3 OF 497 BIOSIS COPYRIGHT 2001 BIOSIS
- Visual and neurobehavioral impairment associated with polychlorinated biphenyls.
- . R-1, hearing, grip strength, simple and choice visual reaction AB. times

problem solving for Culture Fair and digit symbol, recall memory, peg placement, trail making A and B for attention and dexterity and long-term memory were tested. A profile of mood states. . visual fields were often constricted. Scores on Culture Fair, digit symbol, vocabulary and verbal recall were lower. Placement of pegs in a slotted pegboard was slower and trail making A and B took longer. Even embedded memory test scores including. .

- L12 ANSWER 4 OF 497 BIOSIS COPYRIGHT 2001 BIOSIS
- . . electricians referred for shortness of breath also had slowness of AB. response, memory loss, and disordered sleep, all of which suggested neurobehavioral impairment. The hypothesis was that diesel exhaust causes central nervous system impairment. Six electricians worked within enclosed concrete walls and roofs. . . with unexposed men, the 16 in this study had significantly impaired reaction time, balance, blink reflex latency R-1, Culture Fair, peg placement, trail making, and verbal recall. Thirteen men had abnormal visual fields, and 11 had abnormal color confusion indices. Nine men had airways obstruction. The author could not attribute abnormalities to confounding factors or bias. Severe neurobehavioral impairment was associated with exposure to confined diesel exhaust. In additional

studies

of diesel-exposed workers, especially drivers of locomotives and trucks,.

- ANSWER 5 OF 497 BIOSIS COPYRIGHT 2001 BIOSIS L12
- Minimally-invasive debulking of ovarian cancer in the rat pelvis by means of photodynamic therapy using the pegylated photosensitizer PEG -m-THPC.
- Interstitial photodynamic therapy (PDT) using the pegylated AΒ photosensitizer PEG-m-THPC was evaluated as a minimally-invasive procedure to selectively debulk unrespectable pelvic ovarian cancer (NuTu-19) in immunocompetent rats. To assess tumour selectivity, **PEG-**m-THPC at dosages of 0.3, 3.0 and 30 mg kg-1 body weight was administered intravenously to 30 rats 4 weeks following. . . and 200 J cm-1 diffuser-length for 30 mg kg-1 and between 300 and 500 J cm-1 for 3 mg kg-1 PEG-m-THPC. Significant damage to normal pelvic organs was only seen if 30 mg kg-1 photosensitizer was activated with profical doses of. . . for at least 2 weeks and the intestinal and ufinally tract

remained functional. No clinical signs of blood vessel or nerve injury were observed. Mean overall survival of untreated tumour-bearing rats was 25.0 +- 4.5 days compared to 38.4 +- 3.8 days and 40.0 +- 3.6 days for rats treated with 3 mg kg-1 or 9 mg kg-1 **PEG** -m-THPC mediated PDT respectively (P < 0.05). We conclude that **PEG** -m-THPC mediated PDT has a favourable therapeutic window and that this minimally-invasive procedure can reduce pelvic cancer bulks effectively and selectively.

L12 ANSWER 1 OF 497 ADISALERTS COPYRIGHT 2001 (ADIS)

ACCESSION NUMBER: 2000:12131 ADISALERTS

DOCUMENT NUMBER: 800827951

TITLE: A phase I trial of solubilized DR2:MBP84-102 (AG284) in

multiple sclerosis

ADIS TITLE: AG 284: therapeutic use.; Multiple sclerosis;

Phase I trial

AUTHOR: Goodkin D E; Shulman M; Winkelhake J; Waubant E; Andersson

P B; et al

CORPORATE SOURCE: University of California at San Francisco/Mt Zion Multiple

Sclerosis Center, San Francisco, California, USA; Anergen,

Inc., Redwood City, California, USA

SOURCE: Neurology Neurology 54: 1414 1420, 11 Apr 2000. (Apr 11,

2000)

DOCUMENT TYPE: (Clinical study)

REFERENCE: Neurological Disorders (Summary): Alert no. 6, 2000

FILE SEGMENT: Summary LANGUAGE: English WORD COUNT: 918

L12 ANSWER 2 OF 497 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 2001:18472 BIOSIS DOCUMENT NUMBER: PREV200100018472

TITLE: Limitations of cyclosporin A inhibition of the

permeability

transition in CNS mitochondria.

AUTHOR(S): Brustovetsky, Nickolay; Dubinsky, Janet M. (1)

CORPORATE SOURCE: (1) Departments of Neuroscience and Physiology, University

of Minnesota Medical School, 321 Church Street SE, 6-145 Jackson Hall, Minneapolis, MN, 55455: dubin001@tc.umn.edu

USA

SOURCE: Journal of Neuroscience, (November 15, 2000) Vol. 20, No.

22, pp. 8229-8237. print.

ISSN: 0270-6474.

DOCUMENT TYPE: Article LANGUAGE: English SUMMARY LANGUAGE: English

L12 ANSWER 3 OF 497 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 2000:463936 BIOSIS DOCUMENT NUMBER: PREV200000463936

TITLE: Visual and neurobehavioral impairment

associated with polychlorinated biphenyls.

AUTHOR(S): Kilburn, Kaye H. (1)

CORPORATE SOURCE: (1) Environmental Sciences Laboratory, University of

Southern California School of Medicine, 2025 Zonal Avenue,

CSC 201, Los Angeles, CA, 90033 USA

SOURCE: Neurotoxicology (Little Rock), (August, 2000) Vol. 21, No.

4, pp. 489-500. print.

ISSN: 0161-813X.

DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

L12 ANSWER 4 OF 497 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 2000:236489 BIOSIS DOCUMENT NUMBER: PREV200000236489

TITLE: Effects of diesel exhaust on neurobehavioral and pulmonary

functions.

Kilburn, Kaye H. (1) AUTHOR(S):

(1) Environmental Sciences Laboratory, University of CORPORATE SOURCE:

Southern California, School of Medicine, 2025 Zonal

Aveneu,

CSC 201, Los Angeles, CA, 90033 USA

Archives of Environmental Health, (Jan. Feb., 2000) Vol. SOURCE:

55, No. 1, pp. 11-17.

-ISSN: 0003-9896. -

DOCUMENT TYPE:

LANGUAGE:

Article English English

L12 ANSWER 5 OF 497 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 1999:525217 BIOSIS

SUMMARY LANGUAGE:

PREV199900525217

DOCUMENT NUMBER: TITLE:

Minimally-invasive debulking of ovarian cancer in the rat

pelvis by means of photodynamic therapy using the

pegylated

photosensitizer PEG-m-THPC.

AUTHOR(S):

Hornung, R.; Fehr, M. K.; Monti-Frayne, J.; Tromberg, B.

J.; Berns, M. W.; Tadir, Y. (1)

CORPORATE SOURCE:

(1) Beckman Laser Institute and Medical Clinic, University of California Irvine, 1002 Health Sciences Road E, Irvine,

CA, 92612 USA

SOURCE:

British Journal of Cancer, (Oct., 1999) Vol. 81, No. 4,

pp.

631-637.

ISSN: 0007-0920.

DOCUMENT TYPE: LANGUAGE:

Article English

SUMMARY LANGUAGE:

English

=> d his

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FILE 'REGISTRY' ENTERED AT 10:31:16 ON 01 FEB 2001

L15185 S POLYETHYLENE GLYCOL

L2 2535 S POLYPROPYLENE GLYCOL

L3 64 S POLYBUTYLENE GLYCOL

L41 S POLYPENTYLENE GLYCOL L5 O S POLYHEXYLENE GLYCOL

L6 O S POLYHEPTYLENE GLYCOL

L7 O S POLYOCTYLENE GLYCOL

L8 O S POLYNONYLENE GLYCOL L9 O S POLYDECYLENE GLYCOL

> FILE 'ADISALERTS, ADISINSIGHT, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL, DRUGU, DRUGUPDATES, ...' ENTERED AT 10:35:27 ON 01 FEB 2001

445916 S L1 OR L2 OR L3 OR L4 OR PEG OR (POLYALKYLENE (W) GLYCOL?) L10 129303 S ((SPINAL OR SPINE?) OR NEURO? OR NERVE? OR AXON?) (W) L11

(INJURY

497 S L10 AND L11 L12

=> s 112 and (py < 1998)

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<---->
SEARCH ENDED BY USER
=> s py<1998
<---->
SEARCH ENDED BY USER
=> s py>1999
SEARCH ENDED BY USER
=> s PY<1998
<---->
u
SEARCH ENDED BY USER
=> s 112 and (py < 1998)
'1998' NOT A VALID FIELD CODE
   3 FILES SEARCHED...
   6 FILES SEARCHED...
  8 FILES SEARCHED...
 11 FILES SEARCHED...
 13 FILES SEARCHED...
  16 FILES SEARCHED...
'1998' NOT A VALID FIELD CODE
  28 FILES SEARCHED...
'1998' NOT A VALID FIELD CODE
  36 FILES SEARCHED...
'1998' NOT A VALID FIELD CODE
  41 FILES SEARCHED...
  45 FILES SEARCHED...
'1998' NOT A VALID FIELD CODE
  49 FILES SEARCHED...
  52 FILES SEARCHED...
L13
          254 L12 AND (PY < 1998)
=> d ibib ti kwic 1-5
L13 ANSWER 1 OF 254 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER:
                   1995:270403 BIOSIS
DOCUMENT NUMBER:
                   PREV199598284703
TITLE:
                   Gastro-oesophageal reflux and feeding problems after
                   gastrostomy in children with severe neurological
                 impairment.
AUTHOR(S):
                   Heine, R. G.; Reddihough, D. S.; Catto-Smith, A. G. (1)
CORPORATE SOURCE:
                   (1) Dep. Gastroenterol., Royal Child. Hosp., Flemington
                   Rd., Parkville, Victoria 3052 Australia
SOURCE:
                   Developmental Medicine and Child Neurology, (1995) Vol.
37,
                   No. 4, pp. 320-329.
                   ISSN: 0012-1622.
DOCUMENT TYPE:
                   Article
```

LANGUAGE: English

SUMMARY LANGUAGE: English; French; German; Spanish

TI Gastro-oesophageal reflux and feeding problems after gastrostomy in children with severe neurological impairment.

- TI Gastro-oesophageal reflux and feeding problems after gastrostomy in children with severe neurological impairment.
- SO Developmental Medicine and Child Neurology, (1995) Vol. 37, No. 4, pp. 320-329.
 ISSN: 0012-1622.
- This study evaluated the effect of percutaneous endoscopic gastrostomy (
 PEG) on the feeding problems and gastro-oesophageal raflux (GOR) of 30 consecutive children with severe neurological impairment who had PEG between October 1990 and March 1993. Evaluation was by questionnaire, clinical history, examination, 24-hour oesophageal pH monitoring and endoscopy. Gastrostomy. . . severity of GOR was significantly increased in eight patients and fundoplication was required in five. 24-hour oesophageal pH measurements before PEG did not reliably predict subsequently increased GOR. Seven patients died, but their deaths were apparently unrelated to GOR. PEG effectively provides nutrition, improves feed-related stresses, but may exacerbate GOR.

L13 ANSWER 2 OF 254 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 1995:216084 BIOSIS DOCUMENT NUMBER: PREV199598230384

TITLE: Fracture of the odontoid peg in ankylosing

spondylitis: Case report.

AUTHOR(S): Peh, Wilfred C. G. (1); Ho, Eric K. W.

CORPORATE SOURCE: (1) Dep. Diagnostic Radiol., Univ. Hong Kong, Queen Mary

Hosp., Hong Kong Hong Kong

SOURCE: Journal of Trauma, (1995) Vol. 38, No. 3, pp. 361-363.

ISSN: 0022-5282.

DOCUMENT TYPE: Article LANGUAGE: English

TI Fracture of the odontoid **peg** in ankylosing spondylitis: Case report.

TI Fracture of the odontoid **peg** in ankylosing spondylitis: Case report.

SO Journal of Trauma, (1995) Vol. 38, No. 3, pp. 361-363. ISSN: 0022-5282.

AB. . . ankylosing spondylitis tend to affect the lower cervical spine. We describe a 50-year-old man who sustained fractures of the odontoid peg and body of the second cervical vertebra after a hyperextension injury. In absence of atlanto-occipital fusion, deformity from previous lower cervical spine injury may have contributed to susceptibility for this very rare combination of

fractures.

The patient was treated surgically with a good.

L13 ANSWER 3 OF 254 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 1995:182015 BIOSIS DOCUMENT NUMBER: PREV199598196315

TITLE: Neurotoxic effects from residential exposure to chemicals

from an oil reprocessing facility and superfund site.

AUTHOR(S): Kilburn, Kaye H. (1); Warshaw, Raphael H.

CORPORATE SOURCE: (1) Environmental Sciences Lab., Univ. Southern Calif.

Sch.

Med., 2025 Zonal Avenue, CSC 201, Los Angeles, CA 90033

USA

SOURCE: Neurotoxicology and Teratology, (1995) Vol. 17, No. 2, pp.

89-102.

ISSN: 0892-0362.

DOCUMENT TYPE: Article LANGUAGE: English

Neurotoxic effects from residential exposure to chemicals from an oil reprocessing facility and superfund site.

SO Neurotoxicology and Teratology, (1995) Vol. 17, No. 2, pp. 89-102.

ISSN: 0892-0362.

AB. . . Cognitive function in the exposed was impaired as measured by Culture Fair and by block design from the WAIS. Placing pegs in a grooved board and making of trails (A and B) were also impaired Group differences in recall and memory. . . Subjects exposed resident hally for up to 17 years to chemicals dispersed from a waste oil reprocessing plant showed neurophysiological and neuropsychological impairment.

ΙT Miscellaneous Descriptors

> COGNITIVE FUNCTION IMPAIRMENT; DEPRESSION; NEUROPHYSIOLOGICAL IMPAIRMENT; NEUROPSYCHOLOGICAL IMPAIRMENT;

NEUROTOXICOLOGY; VOLATILE ORGANIC CHEMICAL; WASTE OIL REPROCESSING PLANT WORKER

L13 ANSWER 4 OF 254 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 1994:535572 BIOSIS DOCUMENT NUMBER: PREV199497548572

TITLE: Neuronal protection with superoxide dismutase in

repetitive

forebrain ischemia in gerbils.

AUTHOR(S): Truelove, Debbie; Shuaib, Ashfaq (1); Ijaz, Sadiq;

Ishaqzay, Rahmat; Kalra, Jay

CORPORATE SOURCE: (1) Dep. Med., Saskatchewan Stroke Res. Cent., Royal Univ.

Hosp., Saskatoon, SK Canada

SOURCE: Free Radical Biology & Medicine, (1994) Vol. 17, No. 5,

pp.

445-450.

ISSN: 0891-5849.

DOCUMENT TYPE: Article LANGUAGE: English

TI Neuronal protection with superoxide dismutase in repetitive forebrain ischemia in gerbils.

Free Radical Biology & Medicine, (1994) Vol. 17, No. 5, pp. 445-450. SO ISSN: 0891-5849.

AB. severe damage may be secondary to excessive generation of oxygen free radicals. In this study we tested the efficacy of peg -superoxide dismutase (SOD) in a model of repeated ischemia in \P erbils. Superoxide dismutase (SOD) or vehicle (saline) was delivered thatough osmotic. . mu-1), the extent of damage was no different that vehicle-treated controls in the cortex, striatum, and hippocampus Compared to controls, neuronal damage was, however, significantly more severe in the medial geniculate nucleus and the thalamus in the high-dose SOD-treated animals (p lt. . Miscellaneous Descriptors

NEURONAL DAMAGE; OXYGEN FREE RADICAL

L13 ANSWER 5 OF 254 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 1994:226403 BIOSIS DOCUMENT NUMBER: PREV199497239403

TITLE: Identification of myelin basic proteins in circulating

immune complexes associated with lepromatous leprosy.

AUTHOR(S): Corsico, B. (1); Croce, M. V. (1); Mukherjee, R.;

Segal-Eiras, A.

CORPORATE SOURCE: (1) Centro Invest. Immunol. Basicas Aplicadas, Fac.

Ciencias Med., Univ. Nac. de La Plata Argentina

```
Clinical Immunology and Immunopathology, (1994) Vol. 71,
                     No. 1, pp. 38-43.
                     ISSN: 0090-1229.
DOCUMENT TYPE:
                     Article
LANGUAGE:
                     English
TΙ
     Identification of myelin basic proteins in circulating immune complexes
     associated with lepromatous leprosy.
     Clinical Immunology and Immunopathology, (1994) Vol. 71, No. 1, pp.
38-43.
     ISSN: 0090-1229.
AB.
     . . Circulating immune complexes (CIC) were first measured in
     lepromatous patients (LL) by the 125I-C-1q binding assay and the
     polyethylene glycol (PEG) precipitation test. High levels were
     found by both methods (95 and 90% of positives, respectively). LL-CIC
were
     investigated for the presence of neural antigens. CIC were precipitated
in
     3.5% PEG, filtered through protein A-Sepharose affinity
     chromatography, eluted with glycine-HCl, pH 2.8, and washed with PBS;
     fractions after CIC dissociation were studied by SDS-PAGE and Western
     blotting. The LL-CIC PEG precipitates and the glycine-HCl
     eluates were positive in 76 and 71% respectively against anti-myelin
basic
     proteins (MBP) monoclonal antibody, showing. . . an antigen; its
     significance could be related to the pathogenesis of leprosy since the
     liberation of MBP after Mycobacterium leprae nerve
     damage may elicit anti-MBP autoantibodies to myelin breakdown,
     which reacts with peripheral nerve MBP inducing CIC formation. This
     mechanism may be.
ΙT
     Miscellaneous Descriptors
        AUTOANTIBODY; DEMYELINATION; GLIAL FIBRILLARY ACIDIC PROTEIN; NEURAL
        ANTIGEN; NEUROFILAMENT; PATHOGENESIS; PERIPHERAL NERVE
      DAMAGE
=> d his
     (FILE 'HOME' ENTERED AT 10:27:06 ON 01 FEB 2001)
     FILE 'REGISTRY' ENTERED AT 10:31:16 ON 01 FEB 2001
L1
           5185 S POLYETHYLENE GLYCOL
L2
           2535 S POLYPROPYLENE GLYCOL
L3
             64 S POLYBUTYLENE GLYCOL
L4.
              1 S POLYPENTYLENE GLYCOL
L5
              0 S POLYHEXYLENE GLYCOL
L6
              0 S POLYHEPTYLENE GLYCOL
L7
              0 S POLYOCTYLENE GLYCOL
L8
              0 S POLYNONYLENE GLYCOL
L9
              0 S POLYDECYLENE GLYCOL
     FILE 'ADISALERTS, ADISINSIGHT, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS,
     BIOCOMMERCE, BIOSIS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS,
     CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DGENE, DRUGB, DRUGLAUNCH,
     DRUGMONOG2, DRUGNL, DRUGU, DRUGUPDATES, ...' ENTERED AT 10:35:27 ON 01
     FEB 2001
L10
         445916 S L1 OR L2 OR L3 OR L4 OR PEG OR (POLYALKYLENE (W) GLYCOL?)
L11
         129303 S ((SPINAL OR SPINE?) OR NEURO? OR NERVE? OR AXON?) (W)
(INJURY
L12
            497 S L10 AND L11
L13
            254 S L12 AND (PY < 1998)
```

SOURCE:

=> duplicate

ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove

ENTER L# LIST OR (END):113

DUPLICATE IS NOT AVAILABLE IN 'ADISINSIGHT, BIOCOMMERCE, DGENE, DRUGLAUNCH, DRUGMONOG2, DRUGUPDATES, FOREGE, GENBANK, KOSMET, MEDICONF, PHAR, SYNTHLINE'. ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE DUPLICATE PREFERENCE IS 'BIOSIS, BIOTECHDS, BIOTECHNO, CANCERLIT, CAPLUS, DRUGU, EMBASE, ESBIOBASE, HEALSAFE, IFIPAT, JICST-EPLUS, LIFESCI, MEDLINE, NIOSHTIC, PASCAL, PROMT, SCISEARCH, TOXLINE, TOXLIT, USPATFULL, WPIDS' KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n

PROCESSING COMPLETED FOR L13

L14180 DUPLICATE REMOVE L13 (74 DUPLICATES REMOVED)

=> d 175-180 ibib kwic

L14 ANSWER 175 OF 180 NIOSHTIC

ACCESSION NUMBER: 1997:115492 NIOSHTIC

DOCUMENT NUMBER: NIOSH-00157709

TITLE: Neurological Picture Of Organic Solvent Poisoning In

Industry. A Retrospective Clinical Study Of 37 Patients

Juntunen, J.; Hupli, V.; Hernberg, S.; Luisto, M. AUTHOR(S):

SOURCE: International Archives of Occupational and Environmental

Health, Vol. 46, pages 219-231, 41 references .

PUBLICATION DATE: 1980 LANGUAGE: ENGLISH

PY 1980

ΑB . . . solvents were studied retrospectively in workers in Finland. Patients with organic solvent poisoning were selected if they had

received

pneumoencephalography (PEG) in addition to other neurological evaluations. Most had been exposed to mixed solvents. Carbon-disulfide (75-15-0), trichloroethylene (79-0), styrene (100-42-5), toluene. .

exposures. All subjects had subjective symptoms which could be attributed

to disturbances of central nervous function. Many had peripheral neuropathy. PEG changes suggestive of brain atrophy were seen in 63 percent of the subjects. These were slight in 13 of 24.

Nerve damage; Exposure limits; Physiological measurements; Toxic materials; Workers; Environmental factors; Accidents; Toxic effects; Blood sampling; Physiology; Nervous system

L14 ANSWER 176 OF 180 USPATFULL

ACCESSION NUMBER:

80:13074 USPATFULL

TITLE: INVENTOR(S): Transparent radiation penetrable stretcher panel

Rush, Charlie D., Rte. 4, Box 324E, Albany, GA, United

<--

States 31701

NUMBER DATE ______

PATENT INFORMATION: APPLICATION INFO.:

US 4193148 19800318 US 1978-935626 19780821 (5)

DOCUMENT TYPE:

Utility

PRIMARY EXAMINER:

Nunberg, Casmir A.

LEGAL REPRESENTATIVE:

Newton, Hopkins & Ormsby

```
NUMBER OF CLAIMS:
                        9
EXEMPLARY CLAIM:
NUMBER OF DRAWINGS:
                        4 Drawing Figure(s); 3 Drawing Page(s)
LINE COUNT:
                        160
PΙ
       US 4193148 19800318
SUMM
       . . . on still another stretcher to an X-ray department and placed
on
       an X-ray examination table. In the case of some spinal
     injuries and other types of injuries, these movements of the
       patient from one support structure to another can aggravate the injury.
DETD
              by pop rivets 18. Strap receiving slots 15 are preferably
       provided in the patient support panel 12, as shown, and peg
       apertures 14 are also formed through the panel 12, FIG. 2, to receive
     pegs rising from opposite sides of the lower frame 10 by means
       of which the horizontal frame 11 is tiltably and.
L14 ANSWER 177 OF 180 NIOSHTIC
ACCESSION NUMBER:
                    1997:117797 NIOSHTIC
DOCUMENT NUMBER:
                    NIOSH-00159535
TITLE:
                    Behavioral Effects Of The Cholinesterase Inhibitor And
                    Insecticide Carbaryl (Sevin)
AUTHOR(S):
                    Albright, M. E.; Simmel, E. C.
SOURCE:
                    Journal of Biological Psychology, Vol. 21, No. 1, pages
                    25-31, 29 references .
PUBLICATION DATE:
                    Jul 1979
LANGUAGE:
                    ENGLISH
PΥ
     1979
           . activity. At sessions seven and eight the animals received a
AB
     subcutaneous (sc) injection of 1.0 milliliter per kilogram (kg)
     polyethylene-glycol-200 (25322-68-3). At 30 minutes before
     session nine and ten one group of animals received sc injections of 10
     milligrams (mg)/kg carbaryl.
CT
     Laboratory animals; Metabolites; Physiological measurements;
     Neurotoxicity; Psychological reactions; Biological effects; Nerve
     damage; Physiology; Toxicology; Biochemical analysis; Neurological
     system
RN
     63-25-2 (carbaryl)
     25322-68-3 (polyethylene-qlycol-200)
L14 ANSWER 178 OF 180 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER:
                    76071206 EMBASE
DOCUMENT NUMBER:
                    1976071206
TITLE:
                    A case of oligophrenic cerebellolental degeneration
                    associated with vascular hypertension and gynecomastia
                    (Japanese).
AUTHOR:
                    Hayabara T.; Yabuki S.; Ikeda H.; Otsuki S.
CORPORATE SOURCE:
                    Dept. Neuropsychiat., Okayama Univ. Med. Sch., Okayama,
                    Japan
SOURCE:
                    CLIN.NEUROL., (1975) 15/3 (110-115).
                    CODEN: RISHBH
DOCUMENT TYPE:
                    Journal
FILE SEGMENT:
                    032
                            Psychiatry
                    800
                            Neurology and Neurosurgery
                    018
                            Cardiovascular Diseases and Cardiovascular Surgery
                    022
                            Human Genetics
LANGUAGE:
                    Japanese
SO
     CLIN.NEUROL., (1975) 15/3 (110-115).
     CODEN: RISHBH
AΒ
        . . the 4 limbs, pes equinocavus and atrophy of peroneal muscles.
A11
```

deep reflexes were brisk. All sensory modalities were normal. **PEG** disclosed the cerebello pontine atrophy. EMG of the peroneal muscles showed lower motor **neuron damage**, and conduction velocity of tibial nerve was decreased, and/ACTH test and Metopilon test showed hypofunction of hypophyseal and suprarenal gland....

L14 ANSWER 179 OF 180 USPATFULL

ACCESSION NUMBER: 73:17011 USPATFULL TITLE: EDUCATIONAL APPARATUS

INVENTOR(S): Magram, David, 2304 Sherwood St., Pittsburgh, PA,

United States 15217

NUMBER DATE

PATENT INFORMATION: US 3728800 19730424 <--

APPLICATION INFO.: US 1971-180659 19710915 (5)

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Grieb, Wm. H.

LEGAL REPRESENTATIVE: Stein; Arland T.; Wettach; Thomas C.; Yeager; Robert

D.

SUMM

NUMBER OF CLAIMS: 5

NUMBER OF DRAWINGS: 17 Drawing Figure(s); 3 Drawing Page(s)

LINE COUNT: 239

PI US 3728800 19730424 <--

SUMM . . . of teaching language, which is analytical, has not been particularly successful with young children, particularly deaf children or those with neurological impairments, foreign students, etc. These children often require special assistance in learning the language patterns and the traditional techniques are

rarely. . .

. . may be advantageously used with the older group of students. I provide a linkage system which includes a combination of pegs

arranged in a geometrical pattern that fit holes in a complementary pattern in a block to be aligned and fitted. . .

DRWD FIGS. 3 and 4 are perspective views of another embodiment in which pegs are utilized to form the linkage;

DRWD FIG. 6 is a block flow pattern illustrative of the possible arrangements

incorporating **peg** linkages and demonstrating the "no-go"

DRWD FIG. 7 is an end view of block illustrating one type of coding for the creation of a peg linkage system; and

DETD Another embodiment of my invention is shown in FIGS. 3-6 in which the blocks include **pegs** 20 arranged in geometrical patterns to interlock in hole 21 of a complementary or similar pattern. Utilizing a geometrical pattern. . . very large number of possible variations.

increase the flexibility of the system and to eliminate possible errors,

a "no-go" **peg** 22 can be incorporated on the receiving end and complementary receiving holes 23 in the projecting end as in FIGS.. 6. With this arrangement, blocks 36 and 38 cannot be assembled to form the improper combination "I is," since no-go **peg** 22 of block 38 is not provided with a receiving hole in that position on

block

DETD As an aid to the assembly of the blocks, especially for younger children, the center **peg** is preferably larger in size and/or longer in length than the other **pegs**. I have found that is is preferable to include in all geometrical patterns a center **peg** 34 and corresponding receiving hole 35. Even if not larger in size, it

facilitates the assemblage of the blocks.

DETD

. . quadrant coding system. FIG. 7 shows the quadrants I - IV in which letters a-e designate either complementary holes or pegs . FIGS. 8-17 illustrate a number of word forms which can be used. Each vertical array represents four sides of a block. As shown, the letters at the top of each array designates the peg location on the right side of the array and hole designation on the left side. The letter set out in parenthesis designates the location of the no-go

peg and the complementary receiving hole therefor. Thus by taking for example a block having pegs BE-AC(D) it can be combined with a block having AC(D)-BD(E) which in turn can be combined with a block BD(E)-BD(D), etc. to form a sentence. The rectangles 41 of FIGS. 9-7 represent the projections or pegs on the indicated blocks that permit only a 180 degree. rotation of the blocks so marked. The shaded rectangle indicates a. . . connection while the unshaded rectangle represents a female connection. It is thus clear, that by taking blocks having the same peg code and combining it with a block having the same hole code, grammatically correct sentences can be formed.

CLM What is claimed is:

- 3. A device as set forth in claim 1 wherein said projections comprise a plurality of pegs and said openings comprise a plurality of holes for receiving said pegs, each block having a unique number and arrangement of said pegs and holes associated with a grammatical word classification.
- 5. A device as set forth in claim 3 wherein said pegs are arranged in a geometrical pattern and said openings include geometrical patterns complementary to the pattern of a grammatically correct.

L14 ANSWER 180 OF 180 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 74099835 EMBASE

DOCUMENT NUMBER:

1974099835

TITLE:

Divergent nature of gastric mucosal permeability and gastric acid secretion in sick patients with general

surgical and neurosurgical disease.

AUTHOR: CORPORATE SOURCE: Gordon M.J.; Skillman J.J.; Zervas N.T.; Silen W. Dept. Surg., Harvard Med. Sch., Boston, Mass. 02215,

United

States

SOURCE:

Annals of Surgery, (1973) 178/3 (285-294).

CODEN: ANSUA5

DOCUMENT TYPE:

Journal

FILE SEGMENT:

800 Neurology and Neurosurgery

048 Gastroenterology

009 Surgery

LANGUAGE:

English

Annals of Surgery, (1973) 178/3 (285-294).

CODEN: ANSUA5

patients with neurosurgical illness. Gastric mucosal permeability

and gastric acid secretion were estimated by the change in the ratio of .DELTA.(Li'/PEG) and .DELTA.(H+/PEG) respectively. Six of the 13 acutely ill general surgical patients had normal GMP. The

remaining seven general surgical patients had. . . unit of blood transfusion occurred prior to study in ten of the 22 patients. Four of

the

five patients with neurological injury who bled, and all of the five general surgical patients who bled had increased GMP, but the former group also. . . not bleed. A relationship between increased GMP and bleeding from the stomach is strongly supported by these

investigations. Patients with **neurological injury** complicated by hypotension and respiratory failure may develop the same kind of ulceration seen in general surgical patients who bleed.. . .

=> s 110 (1) 111

40 FILES SEARCHED... L15 330 L10 (L) L11

=> duplicate

ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove

ENTER L# LIST OR (END):115

DUPLICATE IS NOT AVAILABLE IN 'ADISINSIGHT, BIOCOMMERCE, DGENE, DRUGLAUNCH, DRUGMONOG2, DRUGUPDATES, FOREGE, GENBANK, KOSMET, MEDICONF, PHAR, SYNTHLINE'. ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE DUPLICATE PREFERENCE IS 'ADISALERTS, BIOSIS, BIOTECHDS, CABA, CANCERLIT, CAPLUS, DRUGU, EMBAL, EMBASE, ESBIOBASE, HEALSAFE, IFIPAT, JICST-EPLUS, LIFESCI, MEDLINE, NIOSHTIC, PASCAL, PROMT, SCISEARCH, TOXLINE, TOXLIT, USPATFULL'

KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n

PROCESSING COMPLETED FOR L15 L16 231 DUPLICATE REMOVE L15 (99 DUPLICATES REMOVED)

=> s 110 (p) 111

PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH FIELD CODE - 'AND' OPERATOR ASSUMED 'L16 (P) L72' PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH FIELD CODE - 'AND' OPERATOR ASSUMED 'L18 (P) L74' PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH FIELD CODE - 'AND' OPERATOR ASSUMED 'L19 (P) L75' PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH FIELD CODE - 'AND' OPERATOR ASSUMED 'L23 (P) L79' PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH FIELD CODE - 'AND' OPERATOR ASSUMED 'L25 (P) L81' PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH FIELD CODE - 'AND' OPERATOR ASSUMED 'L38 (P) L94' PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH FIELD CODE - 'AND' OPERATOR ASSUMED 'L39 (P) L95' PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH FIELD CODE - 'AND' OPERATOR ASSUMED 'L40 (P) L96' PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH FIELD CODE - 'AND' OPERATOR ASSUMED 'L41 (P) L97' <----> User Break----> SEARCH ENDED BY USER

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L1

L2

L3

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FILE 'REGISTRY' ENTERED AT 10:31:16 ON 01 FEB 2001 5185 S POLYETHYLENE GLYCOL 2535 S POLYPROPYLENE GLYCOL 64 S POLYBUTYLENE GLYCOL

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 L5
               O S POLYHEXYLENE GLYCOL
 L6
               0 S POLYHEPTYLENE GLYCOL
 L7
               0 S POLYOCTYLENE GLYCOL
 L8
               0 S POLYNONYLENE GLYCOL
 L9
               0 S POLYDECYLENE GLYCOL
      FILE 'ADISALERTS, ADISINSIGHT, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS,
      BIOCOMMERCE, BIOSIS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS,
      CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DGENE, DRUGB, DRUGLAUNCH,
      DRUGMONOG2, DRUGNL, DRUGU, DRUGUPDATES, ...' ENTERED AT 10:35:27 ON 01
      FEB 2001
L10
          445916 S L1 OR L2 OR L3 OR L4 OR PEG OR (POLYALKYLENE (W) GLYCOL?)
          129303 S ((SPINAL OR SPINE?) OR NEURO? OR NERVE? OR AXON?) (W)
L11
 (INJURY
L12
             497 S L10 AND L11
L13
             254 S L12 AND (PY < 1998)
L14
             180 DUPLICATE REMOVE L13 (74 DUPLICATES REMOVED)
L15
             330 S L10 (L) L11
L16
             231 DUPLICATE REMOVE L15 (99 DUPLICATES REMOVED)
=> s 116 and (py < 1998)
 '1998' NOT A VALID FIELD CODE
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   6 FILES SEARCHED...
   8 FILES SEARCHED...
  11 FILES SEARCHED...
  13 FILES SEARCHED...
  16 FILES SEARCHED...
'1998' NOT A VALID FIELD CODE
  28 FILES SEARCHED...
'1998' NOT A VALID FIELD CODE
  36 FILES SEARCHED...
'1998' NOT A VALID FIELD CODE
  41 FILES SEARCHED...
  45 FILES SEARCHED...
'1998' NOT A VALID FIELD CODE
  49 FILES SEARCHED...
  52 FILES SEARCHED...
           117 L16 AND (PY < 1998)
=> d ti ibib kwic tot
     ANSWER 1 OF 117 BIOSIS COPYRIGHT 2001 BIOSIS
     Gastro-oesophageal reflux and feeding problems after gastrostomy in
     children with severe neurological impairment.
ACCESSION NUMBER: 1995:270403 BIOSIS
DOCUMENT NUMBER:
                    PREV199598284703
TITLE:
                    Gastro-oesophageal reflux and feeding problems after
                    gastrostomy in children with severe neurological
                    impairment.
AUTHOR(S):
                    Heine, R. G.; Reddihough, D. S.; Catto-Smith, A. G. (1)
CORPORATE SOURCE:
                    (1) Dep. Gastroenterol., Royal Child. Hosp., Flemington
                    Rd., Parkville, Victoria 3052 Australia
SOURCE:
                    Developmental Medicine and Child Neurology, (1995) Vol.
37,
                    No. 4, pp. 320-329.
                    ISSN: 0012-1622.
```

DOCUMENT TYPE:

Article

LANGUAGE: English

SUMMARY LANGUAGE: English; French; German; Spanish

Developmental Medicine and Child Neurology, (1995) Vol. 37, No. 4, pp. 320-329.

ISSN: 0012-1622.

This study evaluated the effect of percutaneous endoscopic gastrostomy (AB PEG) on the feeding problems and gastro-oesophageal reflux (GOR) of 30 consecutive children with severe neurological impairment who had PEG between October 1990 and March 1993. Evaluation was by questionnaire, clinical history, examination, 24-hour oesophageal pH monitoring and endoscopy. Gastrostomy. . severity of GOR was significantly increased in eight patients and fundoplication was required in five. 24-hour oesophageal pH measurements before PEG did not reliably predict subsequently increased GOR. Seven patients died, but their deaths were apparently unrelated to GOR. PEG effectively provides nutrition, improves feed-related stresses, but may exacerbate GOR.

L17 ANSWER 2 OF 117 BIOSIS COPYRIGHT 2001 BIOSIS

Fracture of the odontoid peg in ankylosing spondylitis: Case report.

ACCESSION NUMBER: 1995:216084 BIOSIS PREV199598230384 DOCUMENT NUMBER:

TITLE: Fracture of the odontoid peg in ankylosing spondylitis:

Case report.

AUTHOR(S): Peh, Wilfred C. G. (1); Ho, Eric K. W.

CORPORATE SOURCE: (1) Dep. Diagnostic Radiol., Univ. Hong Kong, Queen Mary

Hosp., Hong Kong Hong Kong

SOURCE: Journal of Trauma, (1995) Vol. 38, No. 3, pp. 361-363.

ISSN: 0022-5282.

DOCUMENT TYPE: Article LANGUAGE: English

SO Journal of Trauma, (1995) Vol. 38, No. 3, pp. 361-363.

ISSN: 0022-5282.

AB. . . ankylosing spondylitis tend to affect the lower cervical spine. We describe a 50-year-old man who sustained fractures of the odontoid peg and body of the second cervical vertebra after a hyperextension injury. In absence of atlanto-occipital fusion, deformity from previous lower cervical spine injury may have contributed to susceptibility for this very rare combination fractures.

The patient was treated surgically with a good.

L17 ANSWER 3 OF 117 BIOSIS COPYRIGHT 2001 BIOSIS

Neurotoxic effects from residential exposure to chemicals from an oil reprocessing facility and superfund site.

ACCESSION NUMBER: 1995:182015 BIOSIS DOCUMENT NUMBER: PREV199598196315

TITLE: Neurotoxic effects from residential exposure to chemicals

from an oil reprocessing facility and superfund site.

AUTHOR(S): Kilburn, Kaye H. (1); Warshaw, Raphael H.

CORPORATE SOURCE: (1) Environmental Sciences Lab., Univ. Southern Calif.

Sch.

Med., 2025 Zonal Avenue, CSC 201, Los Angeles, CA 90033

USA

SOURCE: Neurotoxicology and Teratology, (1995) Vol. 17, No. 2, pp.

89-102.

ISSN: 0892-0362.

DOCUMENT TYPE: Article LANGUAGE: English

Neurotoxicology and Teratology, (1995) Vol. 17, No. 2, pp. 89-102.

ISSN: 0892-0362.

. . Cognitive function in the exposed was impaired as measured by AB. Culture Fair and by block design from the WAIS. Placing pegs in a grooved board and making of trails (A and B) were also impaired. Group differences in recall and memory. . . Subjects exposed residentially for up to 17 years to chemicals dispersed from a waste oil reprocessing plant showed neurophysiological and neuropsychological impairment.

ANSWER 4 OF 117 BIOSIS COPYRIGHT 2001 BIOSIS L17

Neuronal protection with superoxide dismutase in repetitive forebrain ischemia in gerbils.

ACCESSION NUMBER: 1994:535572 BIOSIS DOCUMENT NUMBER: PREV199497548572

TITLE: Neuronal protection with superoxide dismutase in

repetitive forebrain ischemia in gerbils.

AUTHOR(S): Truelove, Debbie; Shuaib, Ashfaq (1); Ijaz, Sadiq;

Ishaqzay, Rahmat; Kalra, Jay

CORPORATE SOURCE: (1) Dep. Med., Saskatchewan Stroke Res. Cent., Royal Univ.

Hosp., Saskatoon, SK Canada

SOURCE: Free Radical Biology & Medicine, (1994) Vol. 17, No. 5,

pp.

445 - 450.

ISSN: 0891-5849.

DOCUMENT TYPE: Article LANGUAGE: English

Free Radical Biology & Medicine, (1994) Vol. 17, No. 5, pp. 445-450. ISSN: 0891-5849.

. severe damage may be secondary to excessive generation of oxygen AB. free radicals. In this study we tested the efficacy of peg -superoxide dismutase (SOD) in a model of repeated ischemia in gerbils. Superoxide dismutase (SOD) or vehicle (saline) was delivered through . . mu-l), the extent of damage was no different than vehicle-treated controls in the cortex, striatum, and hippocampus. Compared to controls, neuronal damage was, however, significantly more severe in the medial geniculate nucleus and the thalamus in the high-dose SOD-treated animals (p lt.

L17 ANSWER 5 OF 117 BIOSIS COPYRIGHT 2001 BIOSIS

Identification of myelin basic proteins in circulating immune complexes associated with lepromatous leprosy.

ACCESSION NUMBER: 1994:226403 BIOSIS DOCUMENT NUMBER: PREV199497239403

TITLE: Identification of myelin basic proteins in circulating

immune complexes associated with lepromatous leprosy.

AUTHOR(S): Corsico, B. (1); Croce, M. V. (1); Mukherjee, R.;

Segal-Eiras, A.

CORPORATE SOURCE: (1) Centro Invest. Immunol. Basicas Aplicadas, Fac.

Ciencias Med., Univ. Nac. de La Plata Argentina

SOURCE:

Clinical Immunology and Immunopathology, (1994) Vol. 71, No. 1, pp. 38-43.

ISSN: 0090-1229.

DOCUMENT TYPE: Article LANGUAGE: English

Clinical Immunology and Immunopathology, (1994) Vol. 71, No. 1, pp. 38-43.

ISSN: 0090-1229.

. Circulating immune complexes (CIC) were first measured in lepromatous patients (LL) by the 125I-C-1q binding assay and the polyethylene glycol (PEG) precipitation test. High levels were found by both methods (95 and 90% of positives, respectively). LL-CIC were

investigated for the presence of neural antigens. CIC were precipitated in

3.5% PEG, filtered through protein A-Sepharose affinity chromatography, eluted with glycine-HCl, pH 2.8, and washed with PBS; fractions after CIC dissociation were studied by SDS-PAGE and Western blotting. The LL-CIC **PEG** precipitates and the glycine-HCl eluates were positive in 76 and 71% respectively against anti-myelin basic

proteins (MBP) monoclonal antibody, showing. . . an antigen; its significance could be related to the pathogenesis of leprosy since the liberation of MBP after Mycobacterium leprae nerve damage may elicit anti-MBP autoantibodies to myelin breakdown, which reacts with peripheral nerve MBP inducing CIC formation. This mechanism may be.

L17 ANSWER 6 OF 117 BIOSIS COPYRIGHT 2001 BIOSIS

REDUCING POSTISCHEMIC PARAPLEGIA USING CONJUGATED SUPEROXIDE DISMUTASE.

ACCESSION NUMBER: 1991:345230 BIOSIS

DOCUMENT NUMBER: BA92:44605

TITLE: REDUCING POSTISCHEMIC PARAPLEGIA USING CONJUGATED

SUPEROXIDE DISMUTASE.

AUTHOR(S): AGEE J M; FLANAGAN T; BLACKBOURNE L H; KRON I L; TRIBBLE C

CORPORATE SOURCE: UNIVERSITY VIRGINIA, BOX 181, CHARLOTTESVILLE, VA. 22908.

SOURCE: ANN THORAC SURG, (1991) 51 (6), 911-915.

CODEN: ATHSAK.

FILE SEGMENT: BA; OLD LANGUAGE: English

ANN THORAC SURG, (1991) 51 (6), 911-915.

CODEN: ATHSAK.

AB. . an ischemic spinal cord may be partly responsible for neuronal destruction. We studied the effects of polyethylene glycol-conjugated superoxide dismutase (PEG-SOD), a free radical scavenger, as a way of increasing spinal cord tolerance to ischemia. Thirty rabbits underwent 40 minutes of aortic occlusion (a known model of paraplegia). Ten of these animals received 25,000 U/kg of PEG-SOD 24 hours before aortic occlusion and two additional doses of 10,000 U/kg, one before and one subsequent to spinal ischemia. Ten animals received superoxide dismutase in the same dosages as those receiving ${\ensuremath{{\textbf{PEG}}}}$ -SOD. Ten control animals received placebo. All animals were studied for 96 hours, at which time a final neurological examination was performed

and

the results were recorded. Of the 10 animals treated with PEG -SOD, 2 were completely paralyzed whereas 8 had less (7) or no (1) neurological impairment. Eight of the 10 control animals and 9 of the 10 animals receiving superoxide dismutase were completely paralyzed. None of the control animals or animals receiving superoxide dismutase had a normal neurological examination (p.ltoreq. 0.05). Treatment with PEG-SOD before and during occlusion increased the rabbit spinal cord tolerance to a 40-minute ischemic insult. Scavenging free radicals may lessen.

L17 ANSWER 7 OF 117 BIOSIS COPYRIGHT 2001 BIOSIS

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY AND EARLY MORTALITY.

ACCESSION NUMBER: 1991:117321 BIOSIS

DOCUMENT NUMBER: BA91:64711

TITLE: PERCUTANEOUS ENDOSCOPIC GASTROSTOMY AND EARLY MORTALITY.

AUTHOR(S): CLARKSTON W K; SMITH O J; WALDEN J M

CORPORATE SOURCE: DIV. GASTROENTEROL., ST. LOUIS UNIV. MED. CENTER, 3635

VISTA AT GRAND BLVD., PO BOX 15250, ST. LOUIS, MO.

63110-0250.

SOURCE: SOUTH MED J, (1990) 83 (12), 1433-1436.

CODEN: SMJOAV. ISSN: 0038-4348.

FILE SEGMENT: BA; OLD LANGUAGE: English

SOUTH MED J, (1990) 83 (12), 1433-1436.

CODEN: SMJOAV. ISSN: 0038-4348.

AΒ To assess morbidity, mortality, and benefit associated with percutaneous endoscopic gastronomy (\mathbf{PEG}), we retrospectively studied 42 patients who had had PEG. Mortality was exceptionally high during the first 60 days after PEG (43%), and then stabilized. In nearly half of the cases (20/42) the **PEG** tube was removed during the first 60 days because of either death or improvement. Patients with malignancy had a significantly higher morbidity and 60-day mortality than the neurologically impaired. We concluded that patients should be carefully selected for PEG because early mortality is high; a 60-day trial of soft nasogastric feedings should be considered before PEG, and could reduce by nearly half the number of patients failing to receive long-term benefit; and patients

with

malignancy have significantly greater morbidity and mortality after PEG and may not receive the same advantage from the procedure.

ANSWER 8 OF 117 BIOSIS COPYRIGHT 2001 BIOSIS L17

REPEAT PERCUTANEOUS ENDOSCOPIC GASTROSTOMY PEG AN OUTPATIENT PROCEDURE.

ACCESSION NUMBER: 1991:68408 BIOSIS

DOCUMENT NUMBER: BA91:37068

TITLE: REPEAT PERCUTANEOUS ENDOSCOPIC GASTROSTOMY PEG AN

OUTPATIENT PROCEDURE.

AUTHOR(S): CULLADO M J; SLEZAK F A; PORTER J A

CORPORATE SOURCE: 55 ARCH ST., SUITE 3D, AKRON, OHIO 44304, USA. SURG ENDOSC, (1990) 4 (3), 173-174.

SOURCE:

CODEN: SUREEX.

FILE SEGMENT: BA; OLD LANGUAGE: English

SURG ENDOSC, (1990) 4 (3), 173-174.

CODEN: SUREEX.

Patients who have previously undergone percutaneous endoscopic gastrotomy (PEG) with subsequent PEG removal occasionally require elective repeat PEG. Adhesion of the stomach to the abdominal wall after the original PEG allows repeat PEG to be performed as an outpatient procedure and full-volume tube feeding to be started immediately. Elective repeat PEG was performed in ten patients. Repeat $\overrightarrow{\textbf{PEG}}$ was performed at the site of the original PEG in all cases. Six of the ten repeat PEGs were performed as an outpatient procedure. No complications were attributed to repeat PEG, and full-volume tube feedings were tolerated in all cases when attempted. To obviate the need for repeat PEG, we recommend immediate replacement after inadvertent PEG removal and avoiding elective removal of PEGs used in patients with long-term neurologic impairment for at least 6 months.

ANSWER 9 OF 117 BIOSIS COPYRIGHT 2001 BIOSIS

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY A MEANS OF ENTERAL NUTRITION OF PATIENTS WITH SERIOUS CEREBRAL DYSFUNCTION.

ACCESSION NUMBER: 1988:421010 BIOSIS

DOCUMENT NUMBER: BA86:83622

TITLE: PERCUTANEOUS ENDOSCOPIC GASTROSTOMY A MEANS OF ENTERAL

NUTRITION OF PATIENTS WITH SERIOUS CEREBRAL DYSFUNCTION.

AUTHOR(S): PESCHL L; ZEILINGER M; MUNDA W; PREM H; SCHRAGEL D CORPORATE SOURCE: VOSTAND INTERNEN ABT., KRANKENHAUSES STADT WIEN

FLORIDSDORF, HINAYSGASSE 1, A-1210 WIEN.

SOURCE: WIEN KLIN WOCHENSCHR, (1988) 100 (10), 314-318.

CODEN: WKWOAO. ISSN: 0043-5325.

FILE SEGMENT: BA; OLD LANGUAGE: German

SO WIEN KLIN WOCHENSCHR, (1988) 100 (10), 314-318.

CODEN: WKWOAO. ISSN: 0043-5325.

AB. . . or passive dislocation of the tube into the oesophagus with subsequent aspiration. Although these risks are minimized by percutaneous gastrostomy (PEG), aspiration cannot be completely prevented even when this method of feeding is employed. Enteral nutrition was provided by PEG in 33 patients with different cerebral disorders. PEG was indicated when adequate oral intake of food and fluids proved impossible 8 to 12 days after an acute hypoxaemic. .

aspirated after returning to oral nutrition, whereby feeding was certainly

implicated in 1 patient and probable in the other patient. **PEG** enables adequate enteral nutrition of patients with severe **neurological impairment**. The advantages of **PEG** over parenteral nutrition are fewer complications, lower costs and, above all, its superiority in meeting physiological requirements.

L17 ANSWER 10 OF 117 BIOSIS COPYRIGHT 2001 BIOSIS

TI PERCUTANEOUS ENDOSCOPIC GASTROSTOMY CLINICAL EXPERIENCE AND FOLLOW-UP.

ACCESSION NUMBER: 1988:263088 BIOSIS

DOCUMENT NUMBER: BA86:2332

TITLE: PERCUTANEOUS ENDOSCOPIC GASTROSTOMY CLINICAL EXPERIENCE

AND

FOLLOW-UP.

AUTHOR(S): LLANEZA P P; MENENDEZ A M; ROBERTS R; DUNN G D

CORPORATE SOURCE: DEP. GASTROENTEROL., VA MEDICAL CENT., 1310 24TH AVE. S.,

NASHVILLE, TN 37212-2637.

SOURCE: SOUTH MED J, (1988) 81 (3), 321-324.

CODEN: SMJOAV. ISSN: 0038-4348.

FILE SEGMENT: BA; OLD LANGUAGE: English

SO SOUTH MED J, (1988) 81 (3), 321-324.

CODEN: SMJOAV. ISSN: 0038-4348.

AB. . . with this method of enteral feeding, we conducted a retrospective study and follow-up of 73 patients having percutaneous endoscopic gastrostomy (PEG). In addition we conducted a telephone survey of 42 persons who cared for the PEG tube. The most common indication was neurologic impairment of deglutition. Early and late complications occurred in 12% and 33% of cases, respectively, and were usually minor. Our 30-day survival was 74%. Most patients (77%) maintained their weight with standard tube feedings. Satisfaction with and acceptance of the PEG was almost universal. Patients should be carefully selected, with attention to long-range benefit.

ANSWER 11 OF 117 BIOTECHDS COPYRIGHT 2001 DERWENT INFORMATION LTD Method for human recombinant apolipoprotein-E protein or analog

purification;

addition of neutralized fatty acid to Escherichia coli culture medium.

followed by non-ionic surfactant; useful in lipid metabolism disorder therapy, diagnosis or drug therapy

ACCESSION NUMBER: 1993-03250 BIOTECHDS

TITLE: Method for human recombinant apolipoprotein-E protein or

analog purification;

addition of neutralized fatty acid to Escherichia coli

culture medium, followed by non-ionic surfactant; useful in lipid metabolism disorder therapy, diagnosis or drug

therapy

PATENT ASSIGNEE: Bio-Technol.Gen.

PATENT INFO: WO 9300443 **7 Jan 1993**APPLICATION INFO: WO 1991-US4553 26 Jun 1991
PRIORITY INFO: US 1991-4553 26 Jun 1991

DOCUMENT TYPE: Patent LANGUAGE: English

OTHER SOURCE: WPI: 1993-036388 [04]

PI WO 9300443 **7 Jan 1993**

AB. . . the presence of Mg2+ and beta-hydroxybutyrate to give insoluble ApoE; (3) recovering ApoE by centrifugation; (4) treating with non-ionic surfactant PEG(9-10)p-t-octylphenol to solubilize ApoE; (5) treating (by ultrafiltration or ionexchange or cation-exchange chromatography) the solubilized ApoE to concentrate and purify the. .

(optionally crosslinked with a therapeutic or diagnostic agent); a solution of pure ApoE; therapy of atherosclerosis, autoimmune disease, hypocholesterolemia, hyperlipoproteinemia, neuronal

injury, tumor; diagnosis of LDL receptor defect and tumor growth; and a lipid emulsion containing ApoE for drug delivery and tissue.

L17 ANSWER 12 OF 117 BIOTECHDS COPYRIGHT 2001 DERWENT INFORMATION LTD

TI Early regenerative responses induced by monoclonal antibodies directed against a surface glycoprotein of goldfish retinal ganglion cells; hybridoma construction and monoclonal antibody preparation

ACCESSION NUMBER: 1984-08369 BIOTECHDS

TITLE: Early regenerative responses induced by monoclonal

antibodies

directed against a surface glycoprotein of goldfish retinal

ganglion cells;

hybridoma construction and monoclonal antibody

preparation

AUTHOR: Schwartz M; Eshhar N

LOCATION: Department of Neurobiology, The Weizmann Institute of

Science, Rehovot 76100, Israel. EMBO J.; (1984) 3, 6, 1287-93

SOURCE: EMBO J.,
DOCUMENT TYPE: Journal
LANGUAGE: English

SO EMBO J.; (1984 3, 6, 1287-93

AB. . . injected i.v. into BALB/c mice. A booster injection was given 1 $\,$ mth

later. Fusion was performed in the presence of **polyethylene glycol**. Hybridoma culture supernatants were screened for binding
capacity to mechanically disocciated cells of goldfish retina. 1
Selected clone detected antigen. . . antibody-injected, injured site.
The possible regulatory role of the antigenic glycoprotein in
maintaining

nerve integrity and/or in restoring it following axonal injury, is discussed. (36 ref)

L17 ANSWER 13 OF 117 CANCERLIT

TI Experience with percutaneous endoscopic gastrostomy on an otolaryngology service.

ACCESSION NUMBER: 96107950 CANCERLIT

DOCUMENT NUMBER: 96107950

TITLE: Experience with percutaneous endoscopic gastrostomy on an

otolaryngology service.

AUTHOR: Wilson W R; Hariri S M

CORPORATE SOURCE: George Washington University Medical Center, Washington,

DC

20037, USA. EAR, NOSE, AND THROAT JOURNAL, (1995). Vol. 74, SOURCE: No. 11, pp. 760-2. Journal code: EDF. ISSN: 0145-5613. DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) FILE SEGMENT: MEDL; L LANGUAGE: English OTHER SOURCE: MEDLINE 96107950 ENTRY MONTH: 199603 EAR, NOSE, AND THROAT JOURNAL, (1995). Vol. 74, No. 11, pp. 760-2. Journal code: EDF. ISSN: 0145-5613. Seventy-one patients have undergone percutaneous endoscopic gastrostomy (AΒ PEG) on our otolaryngology service. Most commonly, these were neurologically-impaired (63%) or head and neck cancer (31%) patients. The PEG procedures were done, in almost all instances, in the operating room in conjunction with other indicated ORL-HNS procedures such as. . . one major complication, namely, seeding of the gastrostomy site with squamous cell carcinoma from a hypopharyngeal tumor. We conclude that PEG is a useful addition to the armamentarium of the head and neck surgeon. L17 ANSWER 14 OF 117 CANCERLIT Recording neurological impairment in clinical trials of glioma. ACCESSION NUMBER: 95114638 CANCERLIT DOCUMENT NUMBER: 95114638 Recording neurological impairment in clinical trials of TITLE: glioma. Grant R; Slattery J; Gregor A; Whittle I R AUTHOR: Department of Clinical Neurosciences, Western General CORPORATE SOURCE: Hospital, Edinburgh, Scotland, UK. JOURNAL OF NEURO-ONCOLOGY, (1994). Vol. 19, No. SOURCE: 1, pp. 37-49. Journal code: JCP. ISSN: 0167-594X. DOCUMENT TYPE: (CLINICAL TRIAL) (CONTROLLED CLINICAL TRIAL) Journal; Article; (JOURNAL ARTICLE) MEDL; L; Priority Journals FILE SEGMENT: LANGUAGE: English OTHER SOURCE: MEDLINE 95114638 ENTRY MONTH: 199503 JOURNAL OF NEURO-ONCOLOGY, (1994). Vol. 19, No. 1, pp. 37-49. SO Journal code: JCP. ISSN: 0167-594X. clinical response to treatment in cerebral glioma remain poorly AB defined, but could be made more objective if simple measures of neurological impairments were included in the definitions. We assessed the utility of simple fast previously validated tests of limb impairment (Timed nine hole peg test and 10 meter walk), memory (Williams delayed recall test) and language (Boston Aphasia Severity Rating Scale) in fifty patients with primary brain tumours to see if they could act as a surrogate for neurological impairment. The tests were compared with established measures of

physical disability (Barthel Disability Index [BDI]) and handicap. Timed tests of hand. .

L17 ANSWER 15 OF 117 CAPLUS COPYRIGHT 2001 ACS

Effects of superoxide dismutase administration on ischemic brain injury TI

neonatal rats

ACCESSION NUMBER: 1997:241872 CAPLUS

DOCUMENT NUMBER: 126:301680

TITLE: Effects of superoxide dismutase administration on

ischemic brain injury in neonatal rats

AUTHOR(S): Kotani, Hiromi

CORPORATE SOURCE: Dept. Pediatrics, Kyoto Prefectural Univ. of Med.,

Kyoto, 602, Japan

SOURCE: Kyoto-furitsu Ika Daigaku Zasshi (1997),

106(3), 339-351

CODEN: KFIZAO; ISSN: 0023-6012

PUBLISHER: Kyoto-fu Igaku Shinkokai

DOCUMENT TYPE: Journal LANGUAGE: Japanese

SO Kyoto-furitsu Ika Daigaku Zasshi (1997), 106(3), 339-351

CODEN: KFIZAO; ISSN: 0023-6012

AB The therapeutic efficacy of i.p. administration of superoxide dismutase

(SOD) on neuronal damage in neonatal rat brains

subjected to ischemia was histol. studied. Brain ischemia was induced

for

а

two hours in ten-day-old Wistar rats and the percentage of histol. neuronal damage was evaluated 24 h after reperfusion in three regions, namely neocortex, archeocortex, and thalamus in each hemisphere. A treatment group receiving free human SOD (n=9) [Mn-SOD 30,000 U/kg i.p. (n=6) or CuZn-SOD 20,000 U/kg i.p. (n=4)] had no therapeutic effect on the area of neuronal damage. The percentage was significantly reduced in the right neocortex (p<0.01) and the right thalamus (p< 0.05) of a treatment group receiving polyethylene glycol-conjugated SOD (PEG-SOD) twice at a dose of 5,000 U/kg each (n=6). In a treatment group receiving PEG-SOD at the beginning of ischemia, neuronal damage was remarkably reduced in the right archeocortex (p<0.05) and the bilateral thalami (right: p<0.05, left: p<0.01)[2,000 U/kg (n=8)], in the bilateral thalami (p<0.05)[5,000 U/kg (n=16)], in the right neocortex (p<0.05), right archeocortex (p<0.05), and bilateral thalami (right: p<0.01, left: p<0.05)[10,000 U/kg (n=15)]. When **PEG**-SOD (10,000 U/kg) was administered at the beginning of recirculation (n=16), neuronal damage was significantly less in the right neocortex (p<0.01),

right archeocortex (p<0.001), and bilateral thalami (right: p<0.01, left: p<0.05). Since **PEG-**SOD was more effective when it was administered after ischemia than at the beginning of ischemia, it appears as though SOD is effective on neonatal ischemic brain damage not only as

preventive but also as a therapeutic prepn. for neonatal asphyxia.

L17 ANSWER 16 OF 117 CAPLUS COPYRIGHT 2001 ACS

TI Arachidonic acid metabolism and pathophysiologic aspects of subarachnoid hemorrhage in rats

ACCESSION NUMBER: 1990:233381 CAPLUS

DOCUMENT NUMBER: 112:233381

TITLE: Arachidonic acid metabolism and pathophysiologic

aspects of subarachnoid hemorrhage in rats

AUTHOR(S): Gaetani, Paolo; Marzatico, Fulvio; Rodriguez y Baena,

Riccardo; Pacchiarini, Lucia; Vigano, Teresa; Grignani, Guido; Crivellari, Maria Teresa; Benzi,

Gianni

CORPORATE SOURCE: Dep. Surg., Univ. Pavia, Pavia, I-27100, Italy

SOURCE: Stroke (Dallas) (1990), 21(2), 328-32

CODEN: SJCCA7; ISSN: 0039-2499

DOCUMENT TYPE: Journal LANGUAGE: English

Stroke (Dallas) (1990), 21(2), 328-32 SO CODEN: SJCCA7; ISSN: 0039-2499 35121-78-9, PGI2 41598-07-6, ΙT 363-24-6, PGE2 **34901-14-9**, PGD2 72025-60-6, Leukotriene C4 RL: FORM (Formation, nonpreparative) (formation of, by brain tissue after subarachnoid hemorrhage, eicosanoid metab. and pathogenesis of neuronal impairment in relation to) ANSWER 17 OF 117 DRUGU COPYRIGHT 2001 DERWENT INFORMATION LTD L17 Asphyxial Brain Damage in the Newborn: New Insights into Pathophysiology and Possible Pharmacologic Interventions. ACCESSION NUMBER: 1993-38950 DRUGU BPT Asphyxial Brain Damage in the Newborn: New Insights into Pathophysiology and Possible Pharmacologic Interventions. AUTHOR: Giacoia G P Tulsa, Oklahoma, United States LOCATION: South.Med.J. (86, No. 6, 676-82, 1993) 1 Fig. 1 Tab. 38 Ref. SOURCE: CODEN: SMJOAV ISSN: 0038-4348 6161 S Yale, Tusla, OK 74136, U.S.A. AVAIL. OF DOC.: LANGUAGE: English DOCUMENT TYPE: Journal AB; LA; CT FIELD AVAIL.: FILE SEGMENT: Literature PΥ 1993 . . Possible pharmacologic interventions in perinatal asphyxial brain AB. damage include calcium channel blockers, allopurinol, oxypurinol, excitatory amino acid antagonists (MK-801 (dizocilpine)), PEG -superoxide dismutase, PEG-catalase, lazaroids, and magnesium sulfate. ABEX The basic mechanisms of hypoxic-ischemic neuronal damage are cellular ionic shifts, energy failure, calcium-activated phospholipid degradation, and increased release of excitatory amino acids (EAA). Oxygen free radicals are implicated in postasphyxial CNS injury. Neuronal damage is believed to mediated by release of EAA (L-glutamate and L-aspartate) which cause lesions mainly in areas of high NMDA. . . pigs. Mg2+ blocks NMDA-associated calcium influx in perinatal rats and human neonates. Oxygen free radical scavengers are allopurinol, oxypurinol, polyethylene-glycol (PEG) superoxide dismutase, and PEG -catalase. PEG-superoxide dismutase and allopurinol are currently on clinical trial in adults and infants, respectively. Methylprednisolone was effective in cases of head. ANSWER 18 OF 117 DRUGU COPYRIGHT 2001 DERWENT INFORMATION LTD L17 Diazepam Attenuation of Somatostatin-Induced Motor Disturbances and TΙ Neurotoxicity. ACCESSION NUMBER: 1988-46189 DRUGU Diazepam Attenuation of Somatostatin-Induced Motor TITLE: Disturbances and Neurotoxicity. Balaban C D; Roskoms A J; Severs W B AUTHOR: Hershey, Pennsylvania, United States LOCATION: Brain Res. (458, No. 1, 91-96, 1988) 3 Fig. 15 Ref. SOURCE: ISSN: 0006-8993 CODEN: BRREAP Department of Anatomy, M.S.Hershey Medical Center, The AVAIL. OF DOC.: Pennsylvania State University, Hershey, PA 17033, U.S.A. LANGUAGE: English DOCUMENT TYPE: Journal AB; LA; CT FIELD AVAIL.:

FILE SEGMENT:

1988

Literature

. . cell death was reduced from 4/4 controls to 4/13 DZ-treated rats. The results indicated that DZ affords protection against permanent neurologic damage produced by SRIF. . . Male Sprague-Dawley rats (300-450 g) received i.p. pentobarbital (45 mg/kg) followed by either i.p. DZ (5 mg/kg) or vehicle control (PEG-400 1 ml/kg, Fisher). 15 Min later they received i.c.v. SRIF (40 ug) in 5 ul artificial CSF. Animals were later. . . the presence DZ pretreatment prior to SRIF reduced the of neurotoxicity. Results barrel rotation incidence to 10% (2/20 vs. 6/20 PEG controls). The mortality rate of **PEG**-treated controls was 42% (5/12). This was reduced to 10% (2/20) by DZ-pretreatment. DZ-pretreatment reduced the incidence of neuronal damage. Also the decreased incidence of SRIF-related cell death after DZ appears in correlation with the reduced barrel rotation incidence.. . . L17 ANSWER 19 OF 117 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V. Percutaneous endoscopic gastrostomy without an antireflux procedure in neurologically disabled children. ACCESSION NUMBER: 97025131 EMBASE DOCUMENT NUMBER: 1997025131 Percutaneous endoscopic gastrostomy without an antireflux TITLE: procedure in neurologically disabled children. Borowitz S.M.; Sutphen J.L.; Hutcheson R.L. AUTHOR: Dr. S.M. Borowitz, Department of Pediatrics, Univ. of CORPORATE SOURCE: Virginia Hlth. Sci. Center, Box 386, Charlottesville, VA 22908, United States Clinical Pediatrics, (1997) 36/1 (25-29). SOURCE: Refs: 23 ISSN: 0009-9228 CODEN: CPEDAM United States COUNTRY: Journal; Article DOCUMENT TYPE: FILE SEGMENT: 007 Pediatrics and Pediatric Surgery 800 Neurology and Neurosurgery Gastroenterology 048 LANGUAGE: English SUMMARY LANGUAGE: English Clinical Pediatrics, (1997) 36/1 (25-29). Refs: 23 ISSN: 0009-9228 CODEN: CPEDAM In children with major neurologic impairment, AΒ gastrostomies are often used to alleviate malnutrition and feeding difficulties. There has been a trend toward performing 'protective' antireflux surgery in these children. Nineteen children with major neurologic impairment and feeding failure were prospectively evaluated and followed up after placement of percutaneous endoscopic gastrotomy (PEG) without any antireflux procedure. Mean age at PEG placement was 34 months with mean follow-up of 20.7 months. All parents would recommend PEG to families with disabled children, and if given the chance, 95% would elect PEG again for their child. No child developed choking, gagging, or retching postoperatively. At the time of follow-up, postoperative gastroesophageal reflux. L17 ANSWER 20 OF 117 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

Modification of the 'push' technique for percutaneous endoscopic

Modification of the 'push' technique for percutaneous

endoscopic gastrostomy in infants and children.

gastrostomy in infants and children.

1996081051

ACCESSION NUMBER:
DOCUMENT NUMBER:

TITLE:

96081051 EMBASE

AUTHOR: Robertson F.M.; Crombleholme T.M.; Latchaw L.A.; Jacir

N.N.

CORPORATE SOURCE: Division of General/Thoracic Surgery, Children's Hospital

of Philadelphia, 34th Street and Civic Ctr.

Boulevard, Philadelphia, PA 19104, United States

SOURCE: Journal of the American College of Surgeons, (1996) 182/3

(215-218).

ISSN: 1072-7515 CODEN: JACSEX

COUNTRY:
DOCUMENT TYPE:

United States
Journal; Article

FILE SEGMENT:

007 Pediatrics and Pediatric Surgery

009 Surgery

037 Drug Literature Index

LANGUAGE: SUMMARY LANGUAGE: English English

SO Journal of the American College of Surgeons, (1996) 182/3 (215-218).

ISSN: 1072-7515 CODEN: JACSEX

BACKGROUND: Percutaneous endoscopic gastrostomy (PEG) by the AB 'push' technique avoids pericatheter infection, repeated insertion of the endoscope, potential esophageal injury from the catheter, and the. . modification of the 'push' technique has eliminated this problem. STUDY DESIGN: During a 16-month period, 22 infants and children underwent PEG insertion using our modified 'push' technique These cases were reviewed for patient characteristics including age, weight, indication for the procedure, . . . of the procedure, cost, conversion to open technique, and complications. RESULTS: We have used the modified 'push' technique to place PEG tubes in 20 infants and children aged four weeks to 15 years (mean, 13 months), weighing 2.7 to 36 kg (median, 6.0 kg), indicated for failure to thrive due to cystic fibrosis (n = 3) or neurologic impairment (n=19). These patients have had follow-up examination from nine to 30 months after the procedure. Operative time averaged 15 minutes.. . . successful in 95

procedure. Operative time averaged 15 minutes... successful in Spercent of patients with one failure caused by loss of gastric insufflation when Fogarty balloons failed. All PEGs were used within 24 hours. There were no deaths and no pericatheter infectious. CONCLUSIONS: A simple modification of the 'push' technique of PEG insertion eliminated problems with loss of gastric insufflation

previously

encountered in small infants. The modified 'push' technique is safe, simple,. . .

L17 ANSWER 21 OF 117 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

TI [Percutaneous endoscopic gastrostomy: Experiences in children]. ERFAHRUNGEN MIT DER PERKUTANEN ENDOSKOPISCHEN GASTROSTOMIE IN DER PADIATRIE.

ACCESSION NUMBER:

94086278 EMBASE

DOCUMENT NUMBER: TITLE:

[Percutaneous endoscopic gastrostomy: Experiences in

children].

1994086278

ERFAHRUNGEN MIT DER PERKUTANEN ENDOSKOPISCHEN GASTROSTOMIE

IN DER PADIATRIE.

AUTHOR: Kuster P.

CORPORATE SOURCE: Universitats-Kinderklinik, Hufelandstrasse 55, D-45147

Essen, Germany

SOURCE: Monatsschrift fur Kinderheilkunde, (1994) 142/2 (101-105).

ISSN: 0026-9298 CODEN: MOKIAY

COUNTRY: Germany

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 007 Pediatrics and Pediatric Surgery

048 Gastroenterology

LANGUAGE:

German

English; German SUMMARY LANGUAGE:

Monatsschrift fur Kinderheilkunde, (1994) 142/2 (101-105).

ISSN: 0026-9298 CODEN: MOKIAY

. dialysis. Results: We observed no perioperative complications. AΒ Catheter tract infections occurred in 4 patients and were successfully treated nonoperatively. Two neurologically impaired children developed reflux oesophagitis. On follow up either an isocaloric enteral nutrition was possible or in 6 patients, the weight improved. Two - patients died shortly after PEG placement not due to the procedure. We removed one catheter because of leakage at peritoneal dialysis. One child used tube. . . feeding was possible after percutaneous endoscopic gastrostomy. The procedure was simple and safe. The high incidence of reflux oesophagitis in neurologica/ly impaired children warrants careful follow up.

L17 ANSWER 22 OF 117 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

Impact of nutritional rehabilitation on gastroesophageal reflux in neurologically impaired children.

ACCESSION NUMBER: 94075339 EMBASE

DOCUMENT NUMBER:

1994075339

TITLE:

Impact of nutritional rehabilitation on gastroesophageal

reflux in neurologically impaired children.

AUTHOR:

Lewis D.; Khoshoo V.; Pencharz P.B.; Golladay E.S.

Dept. of Gastroenterology/Nutrition, Children's Hospital, CORPORATE SOURCE:

200 Henry Clay Ave, New Orleans, LA 70118, United States

SOURCE:

Journal of Pediatric Surgery, (1994) 29/2 (167-170). ISSN: 0022-3468 CODEN: JPDSA3

United States

COUNTRY: DOCUMENT TYPE:

Journal; Conference Article

FILE SEGMENT:

007 Pediatrics and Pediatric Surgery

009 Surgery

019 Rehabilitation and Physical Medicine

037 Drug Literature Index

048 Gastroenterology

LANGUAGE:

English

SUMMARY LANGUAGE:

English Journal of Pediatric Surgery, (1994) 29/2 (167-170).

ISSN: 0022-3468 CODEN: JPDSA3

The impact of nutritional rehabilitation on gastroesophageal reflux (GER) AΒ in 10 malnourished neurologically impaired children

(NIC) was studied (mean age, 9.1 .+-. 3.1 years). None of the children

had

an antireflux procedure (ARP), and all were fed exclusively through a percutaneous endoscopic gastrostomy (PEG). Malnutrition was defined as triceps skin fold thickness (TSF) bel $oldsymbol{\delta}$ w the fifth percentile for age and sex. GER was established. . . persistent symptoms) underwent ARP. We conclude that despite accompanying GER, successful nutritional rehabilitation can be achieved in malnourished NIC, using PEG feeding and antireflux medication. Although some NIC with GER may need an ARP or long-term medication, in most malnourished NIC. .

L17 ANSWER 23 OF 117 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

[Percutaneous endoscopic gastrostomy: Indications and techniques]. LA GASTROSTOMIA PERCUTANEO ENDOSCOPICA (GPE). INDICAZIONI E TECHNICHE.

ACCESSION NUMBER:

94025241 EMBASE

DOCUMENT NUMBER:

1994025241

TITLE:

[Percutaneous endoscopic gastrostomy: Indications and

techniques].

LA GASTROSTOMIA PERCUTANEO ENDOSCOPICA (GPE). INDICAZIONI

TECHNICHE. Cosentino F.; Distefano M.; Veroux P.F.; Imme A.; Percolla Ist. Patologia Speciale Chirur. III, Unita di Endoscopia

CORPORATE SOURCE: Digestiva, Universita degli Studi, Catania, Italy

Giornale Italiano di Endoscopia Digestiva, (1993) 16/4

(181-188).

ISSN: 0394-0225 CODEN: GIEDEL

COUNTRY: Italy

AUTHOR:

SOURCE:

DOCUMENT TYPE: Journal; Article 009 FILE SEGMENT: Surgery 014 Radiology

Biophysics, Bioengineering and Medical 027

Instrumentation Gastroenterology 048

LANGUAGE: Italian

Italian; English SUMMARY LANGUAGE:

Giornale Italiano di Endoscopia Digestiva, (1993) 16/4 (181-188).

ISSN: 0394-0225 CODEN: GIEDEL

Percutaneous endoscopic gastrostomy (PEG) was introduçed in 1980 as an alternative procedure to traditional methods for the placement of a gastrostomy feeding tube in patients with inability to swallow secondary to neurological impairment, oropharingeal neoplasms and facial trauma. Several variations on the original technique have been developed in last time. The 'Push' and.

L17 ANSWER 24 OF 117 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

Simplified 'push' technique for percutaneous endoscopic gastrostomy in children.

ACCESSION NUMBER: 93326455 EMBASE

DOCUMENT NUMBER: 1993326455

Simplified 'push' technique for percutaneous endoscopic TITLE:

gastrostomy in children.

Crombleholme T.M.; Jacir N.N.; Lobe T. AUTHOR:

Division of Pediatric Surgery, New England Medical Center, CORPORATE SOURCE:

750 Washington St, Boston, MA 02111, United States

Journal of Pediatric Surgery, (1993) 28/10 (1393-1395). SOURCE:

ISSN: 0022-3468 CODEN: JPDSA3

United States COUNTRY: DOCUMENT TYPE: Journal; Article

Pediatrics and Pediatric Surgery FILE SEGMENT: 007

048 Gastroenterology

LANGUAGE: English English SUMMARY LANGUAGE:

Journal of Pediatric Surgery, (1993) 28/10 (1393-1395).

ISSN: 0022-3468 CODEN: JPDSA3

Percutaneous endoscopic gastrostomy (PEG) by the 'pull' AB

technique is the standard method in pediatric patients. Modifications have

been reported for adults but few in. . . A modified Seldinger technique

is used to insert a 14F acrylic Foley catheter. We have used this technique to place PEG tubes in 8 children age 6 weeks to 17 years (mean, 6 years), for failure to thrive due to cystic fibrosis (3), neurological impairment (4), and undetermined cause (1). Operative time averaged 15 minutes. All PEGs were used within 24 hours. This 'push' technique of PEG insertion is safe, simple, quick, and obviates many of the potential risks inherent in the 'pull' technique. The 'push' technique.

L17 ANSWER 25 OF 117 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

TI A case of oligophrenic cerebellolental degeneration associated with vascular hypertension and gynecomastia (Japanese).

ACCESSION NUMBER: 76071206 EMBASE

DOCUMENT NUMBER: 1976071206

TITLE: A case of oligophrenic cerebellolental degeneration

associated with vascular hypertension and gynecomastia

(Japanese).

AUTHOR: Hayabara T.; Yabuki S.; Ikeda H.; Otsuki S.

CORPORATE SOURCE: Dept. Neuropsychiat., Okayama Univ. Med. Sch., Okayama,

Japan

SOURCE: CLIN.NEUROL., (1975) 15/3 (110-115).

CODEN: RISHBH

DOCUMENT TYPE: Journal

FILE SEGMENT: 032 Psychiatry

008 Neurology and Neurosurgery

018 Cardiovascular Diseases and Cardiovascular Surgery

022 Human Genetics

LANGUAGE: Japanese

SO CLIN.NEUROL., (1975) 15/3 (110-115).

CODEN: RISHBH

AB . . . the 4 limbs, pes equinocavus and atrophy of peroneal muscles.

All

deep reflexes were brisk. All sensory modalities were normal. **PEG** disclosed the cerebello pontine atrophy. EMG of the peroneal muscles showed lower motor **neuron damage**, and conduction velocity of tibial nerve was decreased, and/ACTH test and Metopilon test showed hypofunction of hypophyseal and suprarenal gland.

L17 ANSWER 26 OF 117 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B. V.

TI Divergent nature of gastric mucosal permeability and gastric acid secretion in sick patients with general surgical and neurosurgical disease.

ACCESSION NUMBER: 74099835 EMBASE

DOCUMENT NUMBER: 1974099835

TITLE: Divergent nature of gastric mucosal permeability and

gastric acid secretion in sick patients with general

surgical and neurosurgical disease.

AUTHOR: Gordon M.J.; Skillman J.J.; Zervas N.T.; Silen W. CORPORATE SOURCE: Dept. Surg., Harvard Med. Sch., Boston, Mass. 02215,

United

States

SOURCE: Annals of Surgery, (1973) 178/3 (285-294).

CODEN: ANSUA5

DOCUMENT TYPE: Journal

FILE SEGMENT: 008 Neurology and Neurosurgery

048 Gastroenterology

009 Surgery

LANGUAGE: English

SO Annals of Surgery, (1973) 178/3 (285-294).

CODEN: ANSUA5

AB . . . patients with neurosurgical illness. Gastric mucosal

permeability

and gastric acid secretion were estimated by the change in the ratio of .DELTA.(Li'/PEG) and .DELTA.(H+/PEG) respectively. Six of the 13 acutely ill general surgical patients had normal GMP. The remaining seven general surgical patients had. . . unit of blood transfusion occurred prior to study in ten of the 22 patients. Four of

the

five patients with neurological injury who bled, and all of the five general surgical patients who bled had increased GMP, but the former group also. . . not bleed. A relationship between increased

GMP and bleeding from the stomach is strongly supported by these investigations. Patients with neurological injury complicated by hypotension and respiratory failure may develop the same kind of ulceration seen in general surgical patients who bleed. . .

L17 ANSWER 27 OF 117 IFIPAT COPYRIGHT 2001 IFI

SEAT SUPPORT AND RESTRAINT SYSTEM FOR THE HANDICAPPED

1857973 IFIPAT; IFIUDB; IFICDB

-SEAT SUPPORT AND RESTRAINT SYSTEM FOR THE TITLE:

HANDICAPPED

Bergeron, Timothy J, RD 1, Box 40, Dolgeville, NY, INVENTOR(S):

13329

Unassigned PATENT ASSIGNEE(S): Burr, Edgar S PRIMARY EXAMINER: Lamb, Tonya ASSISTANT EXAMINER:

Heslin & Rothenberg AGENT:

DATE NUMBER

US 4750478 19880614 PATENT INFORMATION:

(CITED IN 004 LATER PATENTS) APPLICATION INFORMATION: US 1986-874032 19860613

EXPIRATION DATE: 13 Jun 2006 FAMILY INFORMATION: US 4750478 19880614 DOCUMENT TYPE: UTILITY; REASSIGNED; EXPIRED; CERTIFICATE OF

CORRECTION

3 Jan 1989 CORRECTION DATE: MECHANICAL FILE SEGMENT:

NUMBER OF CLAIMS: 40

GRAPHICS INFORMATION: 5 Drawing Sheet(s), 10 Figure(s).

US 4750478 19880614 (CITED IN 004 LATER PATENTS) ΡI

. . . 1. A seat support and restraint system capable of reducing ECLM neuromuscular dysfunction and skeletal deformation and facilitating therapy of a neurologically impaired occupant seated thereon, comprising: a contoured chair having molded base and back

portions, said base and back portions forming a. . . ACLM 12. The seat support and restraint system of claim 11, wherein said second end has a plurality of peg receiving bores on one side, and wherein said tray assembly securing means comprises a support bar receiving structure secured to said internal frame, said receiving structure having a second support bar receiving opening and a second spring loaded peg in engagable relation with said peg receiving bores in said second end such that said second center support bar may be secured within said second receiving.

19. The seat support and restraint system of claim 18, wherein said

first

arm has a plurality of peg receiving bores on one side, and wherein said footrest assembly securing means comprises a support bar receiving structure secured to said internal frame, said receiving structure having a first support bar receiving opening and a first spring

loaded peg in engagable relation with said peg receiving bores in said first arm such that said first center support bar

may be secured within said first receiving. . .

- . . An adjustable seat support and restraint system capable of reducing neuromuscular dysfunction and skeletal deformation and facilitating therapy of a neurologically impaired occupant seated thereon, comprising: a contoured, portable chair having base and back portions molded of a resilent foam material, said. . .
- . . . An adjustable seat support and restraint system capable of reducing

neuromuscular dysfunction and skeletal deformation and facilitating therapy of a **neurologically impaired** occupant seated thereon comprising: a contoured chair having molded base and back portions, said base and back portions forming a. . .

L17 ANSWER 28 OF 117 JICST-EPlus COPYRIGHT 2001 JST

TI The Role of Percutaneous Endoscopic gastrostomy for the Enteral

Nutrition.

ACCESSION NUMBER: 950537138 JICST-EPlus

TITLE: The Role of Percutaneous Endoscopic gastrostomy for the

Enteral Nutrition.

AUTHOR: HATTORI KOJI; OGURA YUKI; MINATO YUKIHITO; SHINTANI SHUZO;

SHIIGAI TATSUO

CORPORATE SOURCE: Sogobyointoridekyodobyoin

SOURCE: Nippon Noson Igakkai Zasshi (Journal of the Japanese

Association of Rural Medicine), (1995) vol. 44, no. 1, pp.

13-15. Journal Code: Z0313B (Fig. 1, Tbl. 2, Ref. 10)

ISSN: 0468-2513

PUB. COUNTRY: Japan

DOCUMENT TYPE: Journal; Article

LANGUAGE: Japanese STATUS: New

SO Nippon Noson Igakkai Zasshi (Journal of the Japanese Association of Rural Medicine), (1995) vol. 44, no. 1, pp. 13-15. Journal Code: Z0313B (Fig.

1, Tbl. 2, Ref. 10) ISSN: 0468-2513

We report our experience with percutaneous endoscopic gastrostomy(
PEG) to assess the safety and usefulness of the PEG. We
reviewed 21 cases(mean age, 72 years), including 20 patients with
neurological impairment and one patient with cancer of
the stomach. Though two minor complications(wound infection and bleeding
from the stomach) occurred, wound. . . of these patients died(3 died
from pneumonia, 2 from respiratory failure, and 1 from stomach cancer),
but there were no PEG-related deaths. After PEG
procedure, serum protein, albumin and cholesterol improved significantly.
PEG was not only safe but also effective for the nutritional
support and the 4-year survival rate was 56%. By this method, moreover,
half of the patients could leave hospital and return home. In conclusion,
PEG, is thought to be the procedure of choice for the long-term
enteral nutrition. (author abst.)

L17 ANSWER 29 OF 117 MEDLINE

TI Laparoscopic nissen fundoplication with simultaneous percutaneous endoscopic gastrostomy in children.

ACCESSION NUMBER: 96351004 MEDLINE

DOCUMENT NUMBER: 96351004

TITLE: Laparoscopic nissen fundoplication with simultaneous

percutaneous endoscopic gastrostomy in children.

AUTHOR: Heloury Y; Plattner V; Mirallie E; Gerard P; Lejus C CORPORATE SOURCE: Department of Pediatric Surgery, Hopital M`ere-Enfant,

Quai

PUB. COUNTRY:

Moncousu, 44000, Nantes, France.

SOURCE: SURGICAL ENDOSCOPY, (1996 Aug) 10 (8) 837-41.

Journal code: VBF. ISSN: 0930-2794. GERMANY: Germany, Federal Republic of Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199611

SO SURGICAL ENDOSCOPY, (1996 Aug) 10 (8) 837-41.

Journal code: VBF. ISSN: 0930-2794.

. . The aim of the study was to evaluate the results of laparoscopic AΒ Nissen fundoplication (LNF) with simultaneous percutaneous endoscopic gastrostomy (PEG) in children with gastroesophageal reflux (GER) disease documented by upper gastrointestinal contrast and/or pH monitoring

and/or esophageal endoscopy. METHODS: Fifteen LNF + PEGs were performed in children with pathologic antecedents: ten neurologically impaired children, two ORL

(otorhinolaryngeal) pathologies. Two cases of AIDS, and one neuroblastoma.

In one case, disruption of the fundoplication occurred. . . led to a second LNF with a good clinical result. CONCLUSIONS: In conclusion, it is possible to perform LNF and PEG during the same operative procedure. Short-term results are satisfactory with 14% recurrent GER. Long-term results need to be evaluated.

L17 ANSWER 30 OF 117 NIOSHTIC

Neurobehavioural Effects Of Repeated Occupational Exposure To Toluene And Paint Solvents

1997:109199 NIOSHTIC ACCESSION NUMBER:

DOCUMENT NUMBER:

NIOSH-00152879

Neurobehavioural Effects Of Repeated Occupational Exposure TITLE:

To Toluene And Paint Solvents

Cherry, N.; Hutchins, H.; Pace, T.; Waldron, H. A. AUTHOR(S): British Journal of Industrial Medicine, Vol. 42, No. 5, SOURCE:

pages 291-300, 29 references .

CODEN: BJIMAG

PUBLICATION DATE: May 1985 DOCUMENT TYPE: Journal ENGLISH LANGUAGE:

PΥ 1985

. . . particular focus on the nervous system. Subjects also underwent AΒ several behavioral tests, including visual search, digit symbol, block design, grooved peg board, simple unprepared reaction time, memory, and reading tests. Results were compared with those obtained

from

unexposed workers. No evidence of impaired nerve conduction in the ulnar or median nerves was found, and few clinical signs of neurological damage were apparent. Exposed workers performed less well on tests than did nonexposed workers. Workers exposed to paint solvents scored less. . . the workers. The authors conclude that a positive correlation cannot be made between exposure to toluene or paint solvents and neurobehavioral damage.

L17 ANSWER 31 OF 117 PROMT COPYRIGHT 2001 Gale Group

PEG-SOD Offers Improvement In Head Injury Outcome ΤI

ACCESSION NUMBER:

94:487374 PROMT

TITLE:

PEG-SOD Offers Improvement In Head Injury Outcome

Marketletter, (10 Oct 1994) pp. N/A. SOURCE:

ISSN: 0140-4288.

LANGUAGE:

English 299

WORD COUNT:

FULL TEXT IS AVAILABLE IN THE ALL FORMAT

Marketletter, (10 Oct 1994) pp. N/A. SO

ISSN: 0140-4288.

Patients with severe closed head injury treated with the free-radical AΒ scavenger PEG-SOD (polyethylene glycol-superoxide dismutase) showed an 18% relative improvement in favorable outcome compared to placebo, according to results of a Phase. .

Dr Muizelaar said that while the results did not achieve statistical significance, they suggest that PEG-SOD might be the first drug able to improve the functional outcome of severely head-injured patients. In the randomized, placebo-controlled study of 463 patients, those receiving 10,000 units/kg of PEG-SOD as a single intravenous injection within eight hours of injury had an 18% relative improvement in good and moderate outcome.

He . . . production of a large number of free radicals. There are several possible mechanisms for the action of oxygen free-radicals

including neuronal injury, ischemic neuronal injury and vascular damage leading to vasospasm. Although other superoxide dismutase agents also have free-radical scavenging effects, their clinical value is limited by a short biological half-life. The PEG-SOD conjugation using polyethylene glycol extends its availability in the bloodstream to five or six days.

L17 ANSWER 32 OF 117 PROMT COPYRIGHT 2001 Gale Group

TI PEG-SOD OF BENEFIT IN HEAD INJURY ACCESSION NUMBER: 93:757448 PROMT

TITLE: PEG-SOD OF BENEFIT IN HEAD INJURY SOURCE: Marketletter, (9 Aug 1993) pp. N/A.

ISSN: 0140-4288.

LANGUAGE: English WORD COUNT: 301

FULL TEXT IS AVAILABLE IN THE ALL FORMAT

SO Marketletter, (9 Aug 1993) pp. N/A.

ISSN: 0140-4288.

AB Polyethylene glycol-superoxide dismutase (PEG-SOD: Sterling Winthrop), an oxygen free radical scavenger, may be of use in patients with severe head injury after a single. . .

Although . . . free radical scavenging effect, their clinical value is somewhat limited by their short biological half-life of about six

The **PEG-**SOD product overcomes this limitation through its conjugation with polyethylene glycol, which extends its availability in the bloodstream to five or. . .

In the trial, a two-center, randomized, placebo-controlled study which involved 104 patients, subjects were randomized to receive either placebo or PEG-SOD in doses of 2,000, 5,000 or 10,000 units/kg. Within three months of the injury, 27 of the 104 patients had. . . Laboratory . . . that most oxygen radicals form in the cerebral blood vessel walls, and it is this formation that is affected by PEG-SOD. Professor Young pointed out that there are several possible roles for oxygen free radicals after severe brain injury, including neuronal injury, ischaemic neuronal

injury and vascular damage leading to vasospasm, and one or more of these might be affected by **PEG**-SOD. The Phase III trial is due to begin in October.

THIS IS THE FULL TEXT: Copyright 1993 by Marketletter (Publications). .

L17 ANSWER 33 OF 117 SCISEARCH COPYRIGHT 2001 ISI (R)

TI ATTENUATED NEUROPATHOLOGY BY NILVADIPINE AFTER MIDDLE CEREBRAL-ARTERY OCCLUSION IN RATS

ACCESSION NUMBER: 91:57298 SCISEARCH

THE GENUINE ARTICLE: ET895

TITLE: ATTENUATED NEUROPATHOLOGY BY NILVADIPINE AFTER MIDDLE

CEREBRAL-ARTERY OCCLUSION IN RATS

AUTHOR: KAWAMURA S (Reprint); SHIRASAWA M; FUKASAWA H; YASUI N CORPORATE SOURCE: RES INST BRAIN & BLOOD VESSELS, DEPT SURG NEUROL, 6-10

SENSHU KUBOTA MACHI, AKITA 010, JAPAN (Reprint); RES INST

BRAIN & BLOOD VESSELS, DEPT PATHOL, AKITA, JAPAN

COUNTRY OF AUTHOR: JAPAN

STROKE, (1991) Vol. 22, No. 1, pp. 51-55. SOURCE:

Article; Journal DOCUMENT TYPE:

FILE SEGMENT: LIFE; CLIN LANGUAGE: ENGLISH

REFERENCE COUNT: 27

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

STROKE, (1991) Vol. 22, No. 1, pp. 51-55. SO

. . . nylon suture introduced through the extracranial internal ΑB carotid artery to occlude the left middle cerebral artery. Nilvadipine was dissolved in polyethylene glycol 400. Immediately following occlusion, group 1 rats (n = 10) were treated subcutaneously with vehicle and group 2 and 3. . . were 25.5 +/- 11.6% (NS) and 13.9 $\,$ +/- 9.2% (p < 0.05 different from group 1), respectively. Nilvadipine decreased ischemic neuronal injury in a dose-dependent manner and may be of use in the treatment of cerebral ischemia.

L17 ANSWER 34 OF 117 USPATFULL

Mer receptor activation by gas6

ACCESSION NUMBER: 2001:1757 USPATFULL

Mer receptor activation by gas6 TITLE:

Chen, Jian, Burlingame, CA, United States INVENTOR(S):

> Hammonds, R. Glenn, Berkeley, CA, United States Godowski, Paul J., Burlingame, CA, United States Mark, Melanie R., Burlingame, CA, United States Mather, Jennie P., Millbrae, CA, United States

Li, Ronghao, Millbrae, CA, United States

Genentech, Inc., South San Francisco, CA, United PATENT ASSIGNEE(S):

States

(U.S. corporation)

	NUMBER	DATE	
PATENT INFORMATION:	US 6169070 WO 9628548	20010102 19960919	<
APPLICATION INFO.:	US 1996-628747 WO 1996-US3031	19960417 19960305	(8)
			PCT 371 date PCT 102(e) date

Continuation-in-part of Ser. No. US 1995-438861, filed RELATED APPLN. INFO.:

on 10 May 1995, now abandoned Continuation-in-part of Ser. No. US 1995-412253, filed on 28 Mar 1995, now

patented, Pat. No. US 5580984

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Fitzgerald, David L.

Kresnak, Mark T. Flehr Hohbach Test Albritton & Herbert LEGAL REPRESENTATIVE:

NUMBER OF CLAIMS: 4 EXEMPLARY CLAIM: 1

27 Drawing Figure(s); 17 Drawing Page(s) NUMBER OF DRAWINGS:

LINE COUNT: 2940 US 6169070 20010102 PΙ

WO 9628548 19960919

. . . such as mannitol or sorbitol; salt-forming counterions such as DETD sodium; and/or nonionic surfactants such as Tween, Pluronics or polyethylene glycol (PEG).

. . . such as mannitol or sorbitol; salt-forming counterions such as DETD sodium; and/or nonionic surfactants such as Tween, Pluronics or polyethylene glycol (PEG).

```
DETD
       . . damaged spinal cord in an effort to influence regeneration of
       interrupted central axons, for assisting in the repair of peripheral
     nerve injuries and as alternatives to multiple
       autografts. See Levi et al., J. Neuroscience 14(3):1309-1319 (1994).
The
       use of cell culture techniques. . .
L17 ANSWER 35 OF 117 USPATFULL
       4-substituted piperidine analogs and their use as subtype selective
NMDA
       receptor antagonists
ACCESSION NUMBER:
                         2000:134901 USPATFULL
TITLE:
                         4-substituted piperidine analogs and their use as
                         subtype selective NMDA receptor antagonists
INVENTOR(S):
                         Bigge, Christopher F., Ann Arbor, MI, United States
                         Wright, Jonathan, Ann Arbor, MI, United States
                         Cai, Sui Xiong, Foothill, CA, United States
                         Weber, Eckard, Laguna Beach, CA, United States
                         Woodward, Richard, Aliso Viejo, CA, United States
                         Lan, Nancy C., South Pasadena, CA, United States
                         Zhou, Zhang-Lin, Irvine, CA, United States
                         Keana, John F. W., Eugene, OR, United States
PATENT ASSIGNEE(S):
                         Warner-Lambert Company, Morris Plains, NJ, United
                         States (U.S. corporation)
                         Cocensys, Incorporated, Irvine, CA, United States
(U.S.
                         corporation)
                              NUMBER DATE
PATENT INFORMATION:
                         US 6130234 20001010
                         WO 9723214
                                          19970703
                                                                       <--
                         US 1996-91594 19961220
APPLICATION INFO.:
                         WO 1996-US20766 19961220
                                          19980916 PCT 371 date
                                          19980916 PCT 102(e) date
                                NUMBER
                                             DATE
PRIORITY INFORMATION:
                         US 1995-9192 19951222 (60)
DOCUMENT TYPE:
                         Utility
                         Chang, Ceila
PRIMARY EXAMINER:
LEGAL REPRESENTATIVE:
                        Fitzpatrick, Cella, Harper & Scinto
NUMBER OF CLAIMS:
                         16
EXEMPLARY CLAIM:
                         1
LINE COUNT:
                         2289
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       US 6130234 20001010
WO 9723214 19970703
PΙ
       . . . neurodegenerative disorders such as Parkinson's disease [T. Klockgether, L. Turski, Ann. Neurol. 34, 585-593 (1993)], human immunodeficiency virus (HIV) related neuronal injury
SUMM
       , amyotrophic lateral sclerosis (ALS), Alzheimer's disease [P. T.
       Francis, N. R. Sims, A. W. Procter, D. M. Bowen, J. Neurochem.. .
SUMM
       . . . from a stroke, the compounds of the present invention may be
       administered to ameliorate the immediate ischemia and prevent further
     neuronal damage that may occur from recurrent strokes.
       . . . oil, or synthetic fatty acid esters, for example, ethyl oleate
       or triglycerides or polyethylene glycol-400 (the compounds are soluble
       in PEG-400). Aqueous injection suspensions may contain
```

substances which increase the viscosity of the suspension include, for

example, sodium carboxymethyl cellulose, sorbitol,. . .

L17 ANSWER 36 OF 117 USPATFULL

4-substituted piperidine analogs and their use as subtype selective

receptor antagonists

ACCESSION NUMBER:

2000:128355 USPATFULL

4-substituted piperidine analogs and their use as

subtype selective NMDA receptor antagonists

INVENTOR(S):

Bigge, Christopher F., Ann Arbor, MI, United States Yuen, Po-Wai, Ann Arbor, MI, United States Cai, Sui Xiong, Foothill, CA, United States Weber, Eckard, Laguna Beach, CA, United States Woodward, Richard, Aliso Viejo, CA, United States Lan, Nancy C., South Pasadena, CA, United States

Zhou, Zhang-Lin, Irvine, CA, United States Keana, John F. W., Eugene, OR, United States Guzikowski, Anthony P., Eugene, OR, United States

PATENT ASSIGNEE(S):

Warner-Lambert Company, Morris Plains, NJ, United

States (U.S. corporation)

Cocensys, Incorporated, Irvine, CA, United States

(U.S.

corporation)

NUMBER DATE US 6124323 20000926 PATENT INFORMATION: WO 9723216 19970703 US 1998-91598 19980916 (9) WO 9723216 <--APPLICATION INFO.: WO 1996-US20872 19961220 19980916 PCT 371 date 19980916 PCT 102(e) date

> NUMBER DATE _____ ___

PRIORITY INFORMATION: US 1995-9184 19951222 (60)

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Rotman, Alan L.
ASSISTANT EXAMINER: Desai, Rita

LEGAL REPRESENTATIVE: Fitzpatrick, Cella, Harper & Scinto

NUMBER OF CLAIMS: 22

1

EXEMPLARY CLAIM:

NUMBER OF DRAWINGS: 2 Drawing Figure(s); 1 Drawing Page(s)

LINE COUNT:

5772

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 6124323 20000926 PΙ

WO 9723216 19970703

. from a stroke, the compounds of the present invention may be DETD administered to ameliorate the immediate ischemia and prevent further neuronal damage that may occur from recurrent strokes.

. . . oil, or synthetic fatty acid esters, for example, ethyl cleate or triglycerides or polyethylene glycol-400 (the compounds are soluble in **PEG-400**). Aqueous injection suspensions may contain substances which increase the viscosity of the suspension include, for example, sodium carboxymethyl cellulose, sorbitol,. . .

L17 ANSWER 37 OF 117 USPATFULL

2-substituted piperidine analogs and their use as subtype-selective TΙ NMDA

receptor antagonists

ACCESSION NUMBER:

2000:128349 USPATFULL

2-substituted piperidine analogs and their use as TITLE: subtype-selective NMDA receptor antagonists Bigge, Christopher F., Ann Arbor, MI, United States INVENTOR(S): Keana, John F. W., Eugene, OR, United States Cai, Sui Xiong, Foothill, CA, United States Weber, Eckard, Laguna Beach, CA, United States Woodward, Richard, Aliso Viejo, CA, United States Lan, Nancy C., South Pasadena, CA, United States Guzikowski, Anthony P., Eugene, OR, United States Warner-Lambert Company, Morris Plains, NJ, United PATENT ASSIGNEE(S): States (U.S. corporation) Cocensys, Inc., Irvine, CA, United States (U.S. corporation) NUMBER DATE ______ US 6124317 20000926 PATENT INFORMATION: <.--WO 9723215 US 1998-91593 19981118 APPLICATION INFO.: WO 1996-US20767 19961220 19981118 PCT 371 date 19981118 PCT 102(e) date NUMBER DATE ______ US 1995-9182 19951222 (60) PRIORITY INFORMATION: DOCUMENT TYPE: Utility Chang, Ceila PRIMARY EXAMINER: Fitzpatrick, Cella, Harper & Scinto LEGAL REPRESENTATIVE: 13 NUMBER OF CLAIMS: EXEMPLARY CLAIM: 1600 LINE COUNT: CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 6124317 20000926 WO 9723215 19970703 PΙ . . neurodegenerative disorders such as Parkinson's disease [T. SUMM Klockgether, L. Turski, Ann. Neurol. 34, 585-593 (1993)], human immunodeficiency virus (HIV) related neuronal injury , amyotrophic lateral sclerosis (ALS), Alzheimer's disease [P. T. Francis, N. R. Sims, A. W. Procter, D. M. Bowen, J. Neurochem. from a stroke, the compounds of the present invention may be SUMM administered to ameliorate the immediate ischemia and prevent further neuronal damage that may occur from recurrent strokes. . . . oil, or synthetic fatty acid esters, for example, ethyl oleate SUMM or triglycerides or polyethylene glycol-400 (the compounds are soluble in PEG-400). Aqueous injection suspensions may contain substances which increase the viscosity of the suspension include, for example, sodium carboxymethyl cellulose, sorbitol,. . . L17 ANSWER 38 OF 117 USPATFULL Synthetic catalytic free radical scavengers useful as antioxidants for prevention and therapy of disease ACCESSION NUMBER: 2000:41033 USPATFULL Synthetic catalytic free radical scavengers useful as TITLE: antioxidants for prevention and therapy of disease Malfroy-Camine, Bernard, Arlington, MA, United States INVENTOR(S): Doctrow, Susan Robin, Roslindale, MA, United States Eukarion, Inc., Bedford, MA, United States (U.S. PATENT ASSIGNEE(S):

NUMBER DATE

corporation)

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20000404
PATENT INFORMATION:
                        US 6046188
                                                                      <--
                        WO 9640148
                                          19961219
                        US 1998-973577
                                          19980311
APPLICATION INFO .:
                        WO 1996-US10037
                                          19960606
                                          19980311 PCT 371 date
                                          19980311 PCT 102(e) date
                        Continuation-in-part of Ser. No. US 1995-485489, filed
RELATED APPLN. INFO.:
                        on 7 Jun 1995, now patented, Pat. No. US 5696109
DOCUMENT TYPE:
                        Utility
                        Reamer, James H.
PRIMARY EXAMINER:
                        Townsend & Townsend & Crew LLP
LEGAL REPRESENTATIVE:
NUMBER OF CLAIMS:
                        24
EXEMPLARY CLAIM:
                        1
                        28 Drawing Figure(s); 16 Drawing Page(s)
NUMBER OF DRAWINGS:
LINE COUNT:
                        3405
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       US 6046188 20000404
       WO 9640148 19961219
       . . . of an antioxidant salen-metal complex pharmaceutical
DETD
       composition. In preferred embodiments, the method is used for
       preventing, arresting, or treating (1) neurological
     damage such as Parkinson's disease or anoxia injury, (2) cardiac
       tissue necrosis resulting from cardiac ischemia, (3) autoimmune
       neurodegeneration (e.g., encephalitis), (4) acute lung injury such as
in
       sepsis and endotoxemia, and (5) neuronal damage
       resulting from anoxia (e.g., stroke, drowning, brain surgery) or trauma
       (e.g., concussion or cord shock).
       . . . complex or a cocktail thereof dissolved in an acceptable
DETD
       carrier, preferably an aqueous carrier or organic solvent (e.g., DMSO,
       solvated PEG, etc.). Since many of the salen-Mn complexes of
       the invention are lipophilic, it is preferable to include in the
       carrier. . . hydrophobic vehicle may be used, or that an aqueous
       vehicle comprising a detergent or other lipophilic agent (e.g., Tween,
       NP-40, PEG); alternatively, the antioxidant salen complexes
       may be administered as a suspension in an aqueous carrier, or as an
       emulsion.
       . . . ethoxylated sorbitol, hydroxypropyl sorbitol, polyethylene
DETD
       glycols 200-6000, methoxy polyethylene glycols 350, 550, 750, 2000 and
       5000, poly[ethylene oxide] homopolymers (100,000-5,000,000),
     polyalkylene glycols and derivatives, hexylene glycol
       (2-methyl-2,4-pentanediol), 1,3-butylene glycol, 1,2,6-hexanetriol, ethohexadiol USP (2-ethyl-1,3-hexanediol), C15-C18 vicinal glycol, and
       polyoxypropylene derivatives of trimethylolpropane are. .
DETD
       In Vivo Model of Neuronal Injury
       These results illustrate the protective effects of a Synthetic
DETD
Catalytic
       Scavenger (SCS), C7, in various models of neuronal
     damage. C7 was able to protect neurons from acute early
       manifestations of neuronal damage, such as lipid
       peroxidation and loss of synaptic viability, as well as long-term
       manifestations of neuronal injury, such as neuronal
       loss 7 days after toxin injection.
       In view of the positive effects obtained with peripheral injections of
DETD
       C7 in the in vivo models of neuronal injury, we
       conclude that the complex is stable in vivo and crosses the blood brain
       barrier as well as neuronal membranes.
       The positive effects of C7 in various models of neuronal
```

injury indicate that reactive oxygen species, especially the

superoxide radical, play a significant role in the pathology induced by

ischemia and. . . L17 ANSWER 39 OF 117 USPATFULL Human dorsal tissue affecting factor (noggin) and nucleic acids encoding same 1998:150785 USPATFULL ACCESSION NUMBER: Human dorsal tissue affecting factor (noggin) and nucleic acids encoding same Valenzuela, David M., Franklin Square, NY, United INVENTOR(S): States Ip, Nancy Y., Stamford, CT, United States Cudny, Henryk D., Concord, CA, United States Yancopoulos, George D., Yorktown Heights, NY, United States Harland, Richard M., Moraga, CA, United States Smith, William C., Santa Barbara, CA, United States Lamb, Teresa, New York, NY, United States Knecht, Anne, Berkeley, CA, United States Regeneron Pharmaceuticals, Inc., Tarrytown, NY, United PATENT ASSIGNEE(S): States (U.S. corporation) Regents of University of California, Oakland, CA, United States (U.S. corporation) DATE NUMBER US 5843775 19981201 PATENT INFORMATION: WO 9405791 19940317 <--19950922 US 1995-392935 (8) APPLICATION INFO.: WO 1993-US8326 19930902 19950922 PCT 371 date 19950922 PCT 102(e) date Continuation-in-part of Ser. No. US 1992-957401, filed RELATED APPLN. INFO.: on 6 Oct 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-950410, filed on 23 Sep 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-939954, filed on 3 Sep 1992 DOCUMENT TYPE: Utility PRIMARY EXAMINER: Fitzgerald, David L. Kemmerer, Elizabeth C. ASSISTANT EXAMINER: Cobert, Robert J. Pennie & Edmonds LEGAL REPRESENTATIVE: 23 NUMBER OF CLAIMS: EXEMPLARY CLAIM: 29 Drawing Figure(s); 15 Drawing Page(s) NUMBER OF DRAWINGS: LINE COUNT: 2367 CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5843775 19981201 WO 9405791 19940317 PΤ <--. . . loss of neurons, whether central, peripheral, or motorneurons. DRWD In addition, it may be useful for treating damaged nerve cells, e.g., nerves damaged by traumatic conditions such as burns and wounds, diabetes, kidney dysfunction, and the toxic effects of chemotherapeutics used to treat. sugar alcohols such as mannitol or sorbitol; salt-forming DRWD

counterions such as sodium; and/or nonionic surfactants such as Tween,

L17 ANSWER 40 OF 117 USPATFULL

Pluronics or PEG.

Antibodies to neurotrophic factor-4 (NT-4) 97:123048 USPATFULL

ACCESSION NUMBER:

Antibodies to neurotrophic factor-4 (NT-4) TITLE: Rosenthal, Arnon, Pacifica, CA, United States INVENTOR(S):

Genentech, Inc., South San Francisco, CA, United PATENT ASSIGNEE(S):

(U.S. corporation)

DATE NUMBER _____ PATENT INFORMATION: US.5702906 19971230

US 1995-451947 19950526 (8) APPLICATION INFO.:

Division of Ser. No. US 1995-426419, filed on 19 Apr RELATED APPLN. INFO.:

1995 which is a continuation of Ser. No. US

1993-30013,

filed on 22 Mar 1993, now abandoned which is a

continuation-in-part of Ser. No. US 1991-648482, filed

on 31 Jan 1991, now abandoned which is a

continuation-in-part of Ser. No. US 1990-587707, filed

on 25 Sep 1990, now patented, Pat. No. US 5364769

Utility DOCUMENT TYPE:

PRIMARY EXAMINER: Hutzell, Paula ASSISTANT EXAMINER: Gucker, Stephen

LEGAL REPRESENTATIVE: Torchia, PhD, Timothy E.

NUMBER OF CLAIMS: 10 EXEMPLARY CLAIM:

6 Drawing Figure(s); 6 Drawing Page(s) NUMBER OF DRAWINGS:

LINE COUNT: 2046

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5702906 19971230

. . . loss of neurons, whether central, peripheral, or motorneurons. DETD In addition, it may be useful for treating damaged nerve cells, e.g.,

nerves damaged by traumatic conditions such as burns

and wounds, diabetes, kidney dysfunction, and the toxic effects of

chemotherapeutics used to treat. . .

. . . sugar alcohols such as mannitol or sorbitol; salt-forming counterions such as sodium; and/or nonionic surfactants such as Tween, Pluronics or PEG.

L17 ANSWER 41 OF 117 USPATFULL

Synthetic catalytic free radical scavengers useful as antioxidants for prevention and therapy of disease

ACCESSION NUMBER:

97:115268 USPATFULL

TITLE:

Synthetic catalytic free radical scavengers useful as antioxidants for prevention and therapy of disease Malfroy-Camine, Bernard, Arlington, MA, United States

<--

INVENTOR(S):

Doctrow, Susan Robin, Roslindale, MA, United States

Eukarion, Inc., Bedford, MA, United States (U.S. PATENT ASSIGNEE(S):

corporation)

NUMBER DATE ______

PATENT INFORMATION: APPLICATION INFO.:

US 5696109 19971209 US 1995-485489 19950607 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1995-380731, filed on 26 Jan 1995 which is a continuation-in-part of Ser. No. US 1992-987474, filed on 7 Dec 1992, now patented,

Pat. No. US 5403834

NUMBER DATE _____

PRIORITY INFORMATION:

WO 1993-US11857 19931206

DOCUMENT TYPE:

Utility

```
Jarvis, William R. A.
PRIMARY EXAMINER:
                        Townsend and Townsend and Crew LLP
LEGAL REPRESENTATIVE:
NUMBER OF CLAIMS:
                        14
EXEMPLARY CLAIM:
                        1
                        28 Drawing Figure(s); 19 Drawing Page(s)
NUMBER OF DRAWINGS:
                        3441
LINE COUNT:
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       US 5696109 19971209
PΙ
       . . . of an antioxidant salen-metal complex pharmaceutical
DETD
       composition. In preferred embodiments, the method is used for
       preventing, arresting, or treating (1) neurological
     damage such as Parkinson's disease or anoxia injury, (2) cardiac
       tissue necrosis resulting from cardiac ischemia, (3) autoimmune
       neurodegeneration (e.g., encephalitis), (4) acute lung injury such as
in
       sepsis and endotoxemia, and (5) neuronal damage
       resulting from anoxia (e.g., stroke, drowning, brain surgery) or trauma
       (e.g., concussion or cord shock).
       . . . complex or a cocktail thereof dissolved in an acceptable
DETD
       carrier, preferably an aqueous carrier or organic solvent (e.g., DMSO,
       solvated PEG, etc.). Since many of the salen-Mn complexes of
       the invention are lipophilic, it is preferable to include in the
       carrier. . . hydrophobic vehicle may be used, or that an aqueous
       vehicle comprising a detergent or other lipophilic agent (e.g., Tween,
       NP-40, PEG); alternatively, the antioxidant salen complexes
       may be administered as a suspension in an aqueous carrier, or as an
       emulsion.
       . . . ethoxylated sorbitol, hydroxypropyl sorbitol, polyethylene
DETD
       glycols 200-6000, methoxy polyethylene glycols 350, 550, 750, 2000 and
       5000, poly[ethylene oxide] homopolymers (100,000-5,000,000),
     polyalkylene glycols and derivatives, hexylene glycol
       (2-methyl-2,4-pentanediol), 1,3-butylene glycol, 1,2,6-hexanetriol,
       ethohexadiol USP (2-ethyl-1,3-hexanediol), C15-C18 vicinal glycol, and polyoxypropylene derivatives of trimethylolpropane are. . .
       In vivo model of neuronal injury
DETD
       These results illustrate the protective effects of a Synthetic
DETD
Catalytic
       Scavenger (SCS), C7, in various models of neuronal
     damage. C7 was able to protect neurons from acute early
       manifestations of neuronal damage, such as lipid
       peroxidation and loss of synaptic viability, as well as long-term
       manifestations of neuronal injury, such as neuronal
       loss 7 days after toxin injection.
       In view of the positive effects obtained with peripheral injections of
DETD
       C7 in the in vivo models of neuronal injury, we
       conclude that the complex is stable in vivo and crosses the blood brain
       barrier as well as neuronal membranes.
       The positive effects of C7 in various models of neuronal
DETD
     injury indicate that reactive oxygen species, especially the
       superoxide radical, play a significant role in the pathology induced by
       ischemia and.
L17 ANSWER 42 OF 117 USPATFULL
       Modified anti-ICAM-1 antibodies and their use in the treatment of
        inflammation
                         97:114931 USPATFULL
ACCESSION NUMBER:
                         Modified anti-ICAM-1 antibodies and their use in the
TITLE:
                         treatment of inflammation
                         Faanes, Ronald Bertrand, Pound Ridge, NY, United
INVENTOR(S):
```

McGoff, Paul Edward, Watertown, CT, United States

States

Shirley, Bret Allen, New Milford, CT, United States Scher, David Stuart, Danbury, CT, United States Boehringer Inglehiem Pharmaceuticals, Inc.,

PATENT ASSIGNEE(S): Ridgefield,

CT, United States (U.S. corporation)

DATE NUMBER _____ US 5695760 19971209 <--PATENT INFORMATION: US 1995-427355 19950424 (8) APPLICATION INFO.: Utility DOCUMENT TYPE: Feisee, Lila PRIMARY EXAMINER: ASSISTANT EXAMINER: Johnson, Nancy A. Howrey & Simon; Auerbach, Jeffery I. LEGAL REPRESENTATIVE: NUMBER OF CLAIMS: 31 EXEMPLARY CLAIM: 1,12,24 5 Drawing Figure(s); 5 Drawing Page(s) NUMBER OF DRAWINGS: LINE COUNT: 3085 CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5695760 19971209 PΤ . . . model. Bowes, M. P. et al. (Exper. Neurol. 119:215-219 (1993)) DETD reported that the administration of anti-ICAM-1 antibody could reduce the neurological damages associated with stroke in a rabbit cerebral embolism stroke model. Pavilack, M. A. et al. (Invest. Ophthalmol. Vis. Sci. 35:1896. while retaining the in vivo therapeutic efficacy of the DETD antibody. Preferred modifications include modifying the antibody to contain poly(ethylene) glycol ("PEG") adducts. PEG is mildly hydrophobic material having very high aqueous solubility. Most preferably, the poly(ethylene) glycol-modification reaction is conducted using "activated" PEG derivatives. As used herein, DETD "activated PEG derivatives" are derivatives of PEG bearing electrophilic groups that are reactive toward amines (such as lysines) and other nucleophiles are referred to as "activated PEGs." These PEGs have been used extensively for attachment of DETD PEG to proteins and in liposome formulations (Davis, F. F. et al., U.S. Pat. No. 4,179,337; Rhee, W. et. al., U.S.. . . applications, are the monofunctional polymers which are capped DETD on one end with a methyl ether group (mPEG). Reactions of activated PEGs are free from crosslinking and can result in attachment of multiple strands of the polymer to the target molecule. Choice. . . The N-hydroxysuccinimidyl (or NHS) active esters of PEG DETD succinate (SS-PEG) have been the reagents of choice for attachment of PEG to proteins or peptides in many laboratories. These derivatives react with amino groups on proteins under mild conditions in short. . . `an ester link in its backbone and thus has the property of undergoing relatively rapid hydrolysis in vivo. More stable PEG conjugates can be made by use of the succinimidyl derivative of PEG propionic acid (SPA-PEG), which does not possess the ester linkage. This is also true of the corresponding succinimidyl derivative of carboxymethylated PEG (SCM-PEG) which is even more reactive than the SPA-PEG . SCM-PEG is extremely reactive both toward hydrolysis and aminolysis with an hydrolysis half life of less than one minute at pH. . to result in highly poly(ethylene) glycol-modified enzymes which retain nearly 100% specific activity relative to the native protein.

Presumably, the PEG derivative lifetime limits its reactivity

to lysines at the very surface of the protein where their placement is

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. . . may alternatively be employed. Suitable activated
      monofunctional poly(ethylene) glycols include N-hydroxysuccinimidyl
      active esters of the propionic acid of poly(ethylene) glycol ("SPA-
    PEG"), N-hydroxysuccinimidyl active esters of the succcinate
      poly(ethylene) glycol ("SS-PEG"), N-hydroxysuccinimidyl active
      esters of carboxymethylated poly(ethylene) glycol ("SCM-PEG"),
      N-hydroxysuccinimidyl active esters of the poly(ethylene) glycol dimer
      with lysine ("PEG2-NHS"), poly(ethylene) glycol propionaldehyde ("
    PEG-ALDEHYDE"), or N-hydroxysuccinimidyl derivatives of
      norleucine poly(ethylene) glycol ("PEG-NORLEUCINE").
      The most preferred activated mPEG is an N-hydroxysuccinimidyl
derivative
      of mPEG propionic acid ("SPA-PEG"). Polymerization of mPEG
      monomers results in the production of poly(ethylene) glycol-modified
       ("poly(ethylene) glycol-modified") antibodies, which are the most
      preferred modified.
      Much literature has been devoted to the study of protein modification
DETD
by
      attachment of polyethylene glycol (PEG) of various molecular
      weights ranging from 5K to 40K or higher. As indicated, PEG
      has been reported attached to enzymes, peptides and proteins.
       . . for producing poly(ethylene) glycol-modified antibodies, and
DETD
      disclose that such antibodies exhibit reduced immunogenicity. The
method
       involves the covalent attachment of PEG to trinitrobenzene
       sulfonic acid-available amino groups on the protein molecule.
      Tomasi, T. B. et al. (U.S. Pat. No. 4,732,863) disclose that the
DETD
       immunogenicity of PEG-modified antibody varies with the degree
       of modification, and that it is therefore important to control the
       number of PEG molecules attached to the antibody in order to
       balance the reduced immunoreactivity of the antibodies with the need to
       preserve. . .
       . . . therapeutics. Most of the effort reported has been on the
DETD
       procedures used in these modification studies, the synthesis of the
     PEG derivatives and the end result in animal studies or human
       clinical trials; however, there remains a shortage of analytical
       methods. . . attempts have been made to analyze this heterogeneity
       and no technique has emerged which is suitable for resolution of a
     PEG-protein preparation into individual components.
       . . . glycol-modified versions of these molecules but also allowing
DETD
       the separation of individual modified species varying in the number of
       attached PEG strands. This method takes advantage of the
       partitioning of PEG-modified protein into PEG-rich
       phases which is an effective technique in aqueous polymer two-phase
       separations. This new chromatographic technique works equally well for
       proteins modified with 5K PEG derivatives or those modified
       with PEG of higher molecular weight. The method has been
       developed both as a quantitative analytical tool as well as a
       preparative.
       The conjugation reaction is run with the antibody bound to the sICAM-1
DETD
       column, thus masking the binding site from PEG attachment.
       While both the column and solution methods produce mPEG-anti-ICAM-1
       antibody conjugates that retain binding activity, the column method
       permits.
                7.5, and the column is loaded to capacity with enlimomab (2-5)
DETD
       mg/ml in PBS, pH 7.5). A solution of activated SPA-PEG (5 kD)
       is prepared in one column volume of PBS, pH 7.5. The mPEG solution
       typically contains one milligram of.
       . . . First, the number of species in a preparation varies depending
DETD
       upon the technique of poly(ethylene) glycol-modification used (i.e.
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chemistry of PEG derivative, molecular weight, and number of reactive sites on protein). Furthermore, the resolution of the different analytical techniques varies between. PEG is a mildly hydrophobic material with very high aqueous solubility. It has been used to modify proteins, thereby increasing . . . to a chromatographic support. The immobilized hydrophobic DETD moieties may be selected from a broad range of alkyl and aryl groups. PEG is a preferred immobilized moiety. The hydrophobicity of the moiety increases with increasing alkyl length. The protein is adsorbed . . . poly(ethylene) glycol to enhance partitioning in a DETD pseudo-affinity mode since poly(ethylene) glycol modified proteins are known to preferentially partition into PEG phases in 2-phase separation systems (Walter, H. et al., In: Partitioning in Aqueous Two-Phase Systems, Theory, Methods, Uses and Applications. system as has been demonstrated to work in these two-phase systems. While hydrophobic interactions will predominate, the specific interaction between PEG attached to the protein and the PEG -bonded phase may be strong enough to facilitate chromatographic separation of the native protein from the poly(ethylene) glycol modified protein, in addition to resolving individual PEG-protein adducts. . . . ligand hydrophobicity from hydroxypropyl to pentyl plus phenyl DETD were initially tested. In most cases a satisfactory separation of native from **PEG-**modified protein was not achieved. When highly poly(ethylene) glycol modified samples were chromatographed on certain columns the increased hydrophobicity of the PEG-modified antibody was evident in the increase in retention time relative to native protein. However, these columns failed to separate native. Rainin's Hydropore HIC column was tested for its ability to separate DETD native protein from PEG modified protein. This column has the unique property of incorporating PEG as the hydrophobic ligand on a silica based particle (Hatch, R. G., J. Chromatogr. Sci. 28:210 (1990); Chang, J. et al., J. Chromatogr. 319:396 (1985)). The hypothesis of enhancing the hydrophobic interactions with PEG as a bonded phase was tested using this column. This unique chemistry performed well allowing not only separation of the. . . antibody from the poly(ethylene) glycol-modified species and quantitation of the remaining unmodified antibody but also provided separation of the individual 1-PEG, 2-PEG, 3-PEG modified species. With only slight modifications this technique was scaled up and used to purify large quantities of native-free poly(ethylene). organic layer. Poly(ethylene) glycol groups are then DETD covalently attached to the hydrophilic monolayer to produce a chromatographic material. Poly(ethylene) glycol (PEG) is a weakly hydrophobic neutral polymer. PEG is bonded at a surface density that retains proteins at high salt concentrations through interactions between the bonded phase and. . . preparative separation of unpoly(ethylene) glycol-modified DETD enlimomab from the poly(ethylene) glycol-modified BIRR10. The method takes advantage of the partitioning of the PEG modified

protein into PEG rich phases as has been demonstrated in

```
aqueous polymer two-phase separations. Using this chromatographic
      technique, it is possible to rapidly.
       . . . allows estimation of the relative band areas of each
DETD
      poly(ethylene) glycol-modified species. This procedure estimates
      approximately an average of 5 PEGs per BIRR10 molecule. Laser
      desorption mass spectrometry further corroborates these data.
Analytical
      size exclusion chromatography further characterizes BIRR10. A Superdex.
       . . a 200 kD protein while BIRR10 has an apparent Stokes radius of
540
      kD. The addition of five 5 kD PEGs increases the Stokes radius
      by 2.6 fold.
                from Shearwater Polymers, Inc. (Huntsville, Ala.). All mPEGs
DETD
      used in these experiments were an N-hydroxysuccinimidyl derivative of
      mPEG propionic acid (SPA-PEG) of molecular weight 5 kD (cat
       #M-SPA-5000). This activated mPEG is reactive toward amino groups on
      proteins. The chromatographic resin.
       . . . 7.5, and the column was loaded to capacity with enlimomab (2-5)
DETD
      mg/ml in PBS, pH 7.5). A solution of activated SPA-PEG (5 kD)
      was prepared in one column volume of PBS, pH 7.5. The mPEG solution
       contained one milligram of mPEG.
      Experiments to modify enlimomab with PEG have been performed
DETD
      using the succinimidyl ester of carboxymethylated PEG 5000 MW
       (SCM-PEG) or succinimidyl propionate (SPA-PEG).
       Enlimomab has also been derivatized using the 20,000 MW analogs of
these
       2 derivatives. These and other coupling chemistries have.
       . . to mPEG derivative ranged from 10:1 to 1:10. Thus, in some
DETD
       instances there was an excess of lysines, making the m-PEG the
       limiting reagent (and causing a low degree of modification). At the
       other extreme where there was an excess of m-PEG, the lysines
       were limiting (and a high degree of modification occurred).
       Concentrations of enlimomab protein solutions ranged from 1 mg/ml.
       concentrated diglycine, while it was not necessary to quench the SCM
       reactions due to the short half-life. Excess and unreacted PEG
       was diafiltered at least 3.times. using Amicon 30 kD or 100 kD
       centriprep devices.
DETD
       . . . rapid characterization of poly(ethylene) glycol-modified
       preparations of the monoclonal antibody Enlimomab; and (2) to provide a
       general approach to purify PEG-modified proteins.
       Chromatographic testing was performed using several commercially
       available HIC columns: Synchropak (purchased from Synchrom Inc.,
       Lafayette, Ind.), Hydropore HIC.
DETD
       . . in assessing each HIC column. These conjugates varied in
degree
       of poly(ethylene) glycol-modification, chemistry of coupling and
       molecular weight of PEG derivative used in the coupling
       procedures. The derivatives ranged from mixtures which still had as
much
       as 35% remaining native. . . as determined by other techniques to
       mixtures devoid of native and having poly(ethylene) glycol-modified
       species with as many as 30 PEG-5000 units per antibody
       molecule. This wide range in sample compositions enabled a thorough
       assessment of the separation capability of each.
                                                        . .
       . . . ratios of enlimomab:mPEG respectively) showed a shift to much
DETD
       longer retention times of a single species with increasing ratios of
     PEG. However, there was still no separation of residual native
       enlimomab in these highly poly(ethylene) glycol-modified preparations.
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. . as evidenced by the separation of both native enlimomab and

DETD the poly(ethylene) glycol-modified species and from the resolution of multiple **PEG**-enlimomab adducts. HIC employing the phenyl ligand was capable of revealing some heterogeneity in poly(ethylene) glycol-modified samples of enlimomab. This column. . .

DETD . . . HIC method was conducted. This method employed Rainin's Hydropore column, which is only mildly hydrophobic. The Hydropore column

fortuitously has PEG as its hydrophobic ligand. It was hoped that the mechanism of interaction seen in 2-phase systems which incorporate PEG would hold true in the chromatographic separation, and that the use of this ligand would permit the separation of native protein from PEG modified protein. The initial chromatographic runs were performed under identical conditions to those used on the other HIC columns utilizing. . .

DETD . . . the samples; the least modified sample pool shows significant resolution between the native species and what are apparently 2 different PEG modified species. When this sample was tested on other HIC columns no separation at all was detected, nor was any. . significant change in retention time relative to the native antibody identified. Thus some degree of separation of native enlimomab from PEG-modified adducts has been effected on this column.

DETD . . . samples were tested using the Hydropore HIC column system, there was a dramatic change in the retention behavior of the **PEG** derivatives relative to the native enlimomab. With more highly poly(ethylene) glycol-modified species of enlimomab, such as the SCM-5 KD 1:10. . .

DETD The SPA-20 KD reaction mixture illustrates the difference in hydrophobicity when a single **PEG** strand of 20 KD is attached to enlimomab versus multiple strands of 5 KD. With baseline resolution between the native. . . 20 KD sample corroborated these results. Separation seen previously on the hydroxypropyl column indicated that native enlimomab and 1-20 KD **PEG**-enlimomab were equivalent in hydrophobicity (as they co-eluted) and that the second peak at a retention time of 10.29 minutes was the 2-**PEG** derivative.

DETD TABLE 1

Ratio of Enlimomab to SCM Activated PEG (mg/mg) % Native 10:1 41.2 18.3 5:1 9.9 4:1 3:1 5.4 2:1 0.6 1:1 none detected

DETD . . . column permitted the determination of the percentage of native enlimomab remaining in the reaction as a function of ratio of PEG/enlimomab in the reaction mixture. Selected chromatograms from the SCM-5000 series were obtained. These chromatograms illustrate the changes in chromatography seen. . . peaks was obtained from non-reduced SDS-PAGE. The SCM-5000 10:1 sample separated in such SDS-PAGE into the native enlimomab, a major 1-PEG species and a minor 2-PEG species. Several other samples from the SCM-5000 series were also electrophoresed in the same manner with a corresponding

correlation between. . .

DETD . . . case with the Hydropore HIC packing material. There was modest improvement seen in the resolution between native enlimomab and the 1
PEG species as well as improvement in separation between the

individual 1-PEG, 2-PEG, 3-PEG etc. species. The general trend was an overall increase in peak sharpness in going from the 12 to the 5. in many cases with the 12 .mu.m particle material depending on DETD the the poly(ethylene) glycol-modification chemistry, molecular weight of the PEG derivative and the hydrophobicity of the native protein relative to its poly(ethylene) glycol-modified derivatives. Even though the 5 .mu.m particle. . . . separating native protein from poly(ethylene) glycol-modified DETD species, the time course of a poly(ethylene) glycol-modification reaction was monitored using the Ald-5000 PEG derivative coupling to a Fab of another antibody. This reaction was followed over 24 hour period. The Fab was. . . 7.14 minutes versus 9.54 minutes (15 min. linear gradient) and that the poly(ethylene) glycol-modified Fab, with a single 5000 MW $\ensuremath{\text{PEG}}$ attached was significantly more hydrophobic than the native molecule eluting at 9.70 minutes. In this case 1-PEG-Fab species could be readily separated from the native Fab and the quantiation of residual native protein was straightforwared as baseline. . . . as seen in the 60 minute gradient, 30 minutes in run time was DETD saved. With this improved higher resolution method enlimomab-PEG reaction mixtures were then fractionated into individual 1-PEG , 2-PEG, 3-PEG, etc. species. These now homogeneous poly(ethylene) glycol-modified adducts were thoroughly analyzed for binding activity using a competitive specific ELISA (relative. the individual species for further characterization. Hydropore DETD was used analytically for characterizing the poly(ethylene) glycol-modification process, for quantitation of native, 1-PEG , 2-PEG, 3-PEG etc. species and also for the purification of poly(ethylene) glycol-modified-enlimomab on a larger scale (up to 500 mg). Samples which are highly poly(ethylene) glycol-modified were found to DETD be free from residual native PEG as determined by chromatograms and electropherograms. In addition to being able to quantitate native antibody in poly(ethylene) glycol-modified samples this. . . It has been possible to effect an even greater degree of separation DETD between the native antibody and PEG-derivatized species via the incorporation of a isocratic salt hold step at an appropriate point in the gradient. Just at the. . . enlimomab species. Further method development led to a DETD procedure which improves baseline resolution of native enlimomab from the composite of PEG.sub.n -enlimomab adducts. . . with an isocratic hold step positioned at a salt concentration DETD which allows maximum separation of native unpoly(ethylene) glycol-modified enlimomab from PEG.sub.n -enlimomab adducts. The method comprises the following steps: . preparation was injected onto the Poros PE column. enlimomab DETD elutes during the 0.9M hold step and is clearly separated from PEG-enlimomab which is eluted during the second gradient between 0.9M and 0.5M ammonium sulfate. This optimized method developed on an analytical. . run on the 62 ml Poros 20 PE column revealed that enlimomab DETD eluted during the initial isocratic hold step and PEG-modified enlimomab adducts eluted as a single peak during the second gradient portion of the method.

A coomassie-stained non-reduced SDS-PAGE gel of the eluted materials

indicated that a small amount of mono-PEG-enlimomab eluted

DETD

during the isocratic hold step (in which the native enlimomab elutes). It was possible to obtain fractions that consisted of **PEG** -modified enlimomab adducts with no residual non-poly(ethylene) glycol-modified native enlimomab. Thus the scaleup to a larger column format with Poros 20. . .

DETD . . . relative band areas of each poly(ethylene) glycol-modified species of BIRR10. Reduced SDS-PAGE gels provide information on location

and distribution of **PEG** strands on the heavy and light chains of enlimomab.

DETD . . . fairly reproducible and each reaction produced an mPEG-enlimomab conjugate with a similar degree of poly(ethylene) glycol-modification (i.e., average of 5 PEG 5 kD adducts). For each poly(ethylene) glycol-modification run, the enlimomab breakthrough during the load phase and the material eluted from. .

DETD TABLE 9

RR1/ Average Degree

1.1.1:PEG5000

Reaction of **PEG** Activity in slCAM-Ratio Time pH Modification

1 assay

Solution	Met	thod	of form	iing	PEG-Adducts	
1:1	1	hr	6.0	1	Not done	
1:1	1	hr	7.5	1	78%	
1:1	1	hr	8.0	2	58%	
1:1	1	hr	8.5	3.	6	21%
1:4	24	hr	7.5	6	O %	
1:2	4	hr	7.5	4	28%	
1:2	24	hr	7.5	4	36%	
Column Me	eth	to be	formir	ng Pl	EG- Adducts	
1:1			7.5	5	39%	

DETD	TABLE	10	

Experimental Series I

Solution-Based Modification IC.sub.50 .mu.g/ml

Enlimomab	0.2	_
mg Enlimomab:mg PEG		
1:1	0.2	
1:2	3.0	
1:3	1.5	
1:4	<0.1	
10:1	0.4	

Experimental Series II

Solution-Based Modification IC.sub.50 .mu.g/ml

Enlimomab	1.5	
mg Enlimomab:mg PEG		
4:1	6.0	
10:1	1.5	
20:1	0.8	
50:1	0.8	
100:1	1.5	

Experimental Series III

Solution-Based Modification IC.sub.50 .mu.g/ml

```
0.1
Enlimomab
mg Enlimomab:mg PEG
                   Time
                   30 Min. 0.8
10:1
                    1 Hour 0.4
10:1
                    2 Hours 0.4
10:1
                   30 Min. 0.2
20:1
20:1
                    1 Hour 0.4
                    2 Hour 0.4
20:1
 4:1.
            . of homotypic aggregation using 10 .mu.g/ml of the indicated
DETD
       antibody. The solution poly(ethylene) glycol-modified enlimomab had an
       average of 2-3 PEG adducts. The column poly(ethylene)
       glycol-modified enlimomab had an average of 5 PEG adducts.
                     TABLE 11
DETD
```

PEG	_	uirrel nkey	Cynom Monke	nolgus	
Antibody A	intibody	1	2	1	1
No Antibod	ly				
-	· -	3.0+	3.0+	4.0+	4.0+
(Control)					
Enlimomab					
0)	1.0+	0.0+	2.0+	3.0+
Solution F	EG-				
2	2-3	1.0+	1.0+	4.0+	4.0+
Modified					
Enlimomab					
Column PEG	}-				
5)	0.5+	1.5+	3.0+	3.0+
Modified					
Enlimomab					
(BIRR10)					

DETD . . . would be expected that under the rosetting conditions used that

FcRI binding would dominate. The results indicate that attachment of PEG to BIRR10 decreased FcR binding.

DETD . . . 2 shows the anti-enlimomab sera titer (in thousands) of individual rabbits after intraperitoneal injection of either enlimomab, antibody 2183-66M (a PEG-enlimomab derivative having an average of 5 PEG adducts per antibody molecule that was obtained using a CA3 anti-idiotypic column), or BIRR10 (a PEG-enlimomab derivative having an average of 5 PEG adducts per antibody molecule that was otained using an sICAM-1 column). The Figure demonstrates that the PEG-modified enlimomab had substantially lower immunogenicity than the native antibody.

DETD . . . shows the anti-enlimomab sera titer (in thousands) of individual rabbits after intraperitoneal injection of either enlimomab or antibody 930329/4:1 (a PEG-enlimomab derivative having an average of 3 PEG adducts per antibody molecule). This Figure also demonstrates that the PEG-modified enlimomab had substantially lower immunogenicity than the native antibody.

DETD . . . shows the anti-enlimomab sera titer (in thousands) of individual rabbits after intravenous injection of either enlimomab or antibody 1924-11 (a PEG-enlimomab derivative having an average of 2 PEG adducts per antibody molecule). As indicated in the Figure the PEG-modified enlimomab had substantially lower immunogenicity than the native antibody.

DETD TABLE 13

		^7			_
Sera Titers			CEM	m	מנול מינו
Compound I	Mean	Std Dev	SEM	Т	P value
Enlimomab	5.55	0.627	0.313		_
PEG-Enlimomal	0				
	4.50	0.549	0.274	2.52	28 0.045
(made on CA3	anti-				
idiotypic co.					
PEG-Enlimomal					
	4.88	0.995	0.497	1.15	51 0.293
BIRR10					
(column method	od				
PEG-enlimomal	b)				
Enlimomab	4.12	0.620	0.310		
PEG-Enlimoma	b				
	3.11	0.488	0.244	2.5	58 0.043
BIRR10					
(solution me	thod				
PEG-enlimoma	b)				
			4		_
DETD		TABLE	14		
Sera Titers	at Dav	28 and 5	6		_
	ean	Std Dev		T	P value
•					_
Day 28					
Enlimomab 4	.09	0.284	0.142		
PEG -Enlimoma	b				
-	.04	0.404	0.202	4.2	6 0.005
(solution me	thod)				
Day 56					
	.77	0.874	0.437		
PEG-Enlimoma				2 0	- 0 017
	.07	0.569	0.284	3.2	5 0.017
(solution me	thod)				
DETD			. 10.	sup.	7
mAb	No FM	LP	FM		
Treatment		I Expt.			I
		<u> </u>		-	Expt. II
					_
PMBC					_
No mAb	2.8	1.5	3.		3.3
Enlimomab	3.7	2.2	4.	7	4.9
2-PEG-Enlimo	mab				
	3.0	2.4	4.	1	3.0
5- PEG- Enlimo	mab				
	3.2	2.4	4.	0	3.3
(BIRR10)					
Granulocytes		- .	_	_	07.0
No mAb	1.8	3.1			27.0
Enlimomab	2.4	0.9	10	. 5	1/.6
2- PEG- Enlimo			_	^	0. 6
5 ppg 5 11	1.8	1.2	6.	9	9.6
5-PEG-Enlimo		1 0	-	2	7 1
(DIDD10)	2.0	1.3	5.	3	7.4
(BIRR10)					
					

Methods of treating impotency with ciliary neurotrophic factor 97:109873 USPATFULL ACCESSION NUMBER: Methods of treating impotency with ciliary TITLE: neurotrophic Russell, Deborah A., Thousand Oaks, CA, United States INVENTOR(S): Amgen Inc., Thousand Oaks, CA, United States (U.S. PATENT ASSIGNEE(S): corporation) DATE NUMBER US 5691313 19971125 US 1996-704479 19960826 (8) PATENT INFORMATION: APPLICATION INFO.: Continuation of Ser. No. US 1994-298442, filed on 29 RELATED APPLN. INFO.: Aug 1994, now abandoned which is a continuation-in-part of Ser. No. US 1991-735538, filed on 23 Jul 1991, now abandoned DOCUMENT TYPE: Utility Allen, Marianne P. PRIMARY EXAMINER: Levy, Ron K.; Odre, Steven M. LEGAL REPRESENTATIVE: NUMBER OF CLAIMS: EXEMPLARY CLAIM: LINE COUNT: 393 CAS INDEXING IS AVAILABLE FOR THIS PATENT. <--US 5691313 19971125 . . . established, CNTF appears to be released upon injury to the SUMM nervous system and may limit the extent of injury or neuronal damage. . . . described in the '176 application. In a further embodiment, SUMM CNTF is modified by attachment of one or more polyethylene glycol (PEG) or other repeating polymeric moieties. L17 ANSWER 44 OF 117 USPATFULL DNA encoding a tissue differentiation affecting factor 97:96971 USPATFULL ACCESSION NUMBER: DNA encoding a tissue differentiation affecting factor TITLE: De Robertis, Edward M., Pacific Palisades, CA, United INVENTOR(S): States Sasai, Yoshiki, Los Angeles, CA, United States The Regents of the University of California, Oakland, PATENT ASSIGNEE(S): CA, United States (U.S. corporation) NUMBER DATE _____ US 5679783 19971021 <--PATENT INFORMATION: US 1994-343760 19941122 (8) APPLICATION INFO.: Utility DOCUMENT TYPE: PRIMARY EXAMINER: Ulm, John ASSISTANT EXAMINER: Mertz, Prema LEGAL REPRESENTATIVE: Majestic, Parsons, Siebert & Hsue NUMBER OF CLAIMS: 8 EXEMPLARY CLAIM: 1 12 Drawing Figure(s); 12 Drawing Page(s) NUMBER OF DRAWINGS: LINE COUNT: 1285 CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5679783 19971021 . . . loss of neurons, whether central, peripheral, or motorneurons. DETD In addition, it may be useful for treating damaged nerve cells, e.g.,

nerves damaged by traumatic conditions such as burns

and wounds, diabetes, kidney dysfunction, and the toxic effects of

chemotherapeutics used to treat. sugar alcohols such as mannitol or sorbitol; salt-forming DETD counterions such as sodium; and/or nonionic surfactants such as Tween, Pluronics or PEG. L17 ANSWER 45 OF 117 USPATFULL Dorsal tissue affecting factor (noggin) and compositions comprising same ACCESSION NUMBER: 97:86589 USPATFULL Dorsal tissue affecting factor (noggin) and TITLE: compositions comprising same Harland, Richard M., Berkeley, CA, United States INVENTOR(S): Smith, William C., Oakland, CA, United States The Regents of the University of California, Oakland, PATENT ASSIGNEE(S): CA, United States (U.S. corporation) DATE NUMBER _____ US 5670481 19970923 PATENT INFORMATION: US 1994-297633 19940829 (8) APPLICATION INFO.: Continuation of Ser. No. US 1992-939954, filed on 3 RELATED APPLN. INFO.: 1992, now abandoned DOCUMENT TYPE: Utility PRIMARY EXAMINER: Jagannathan, Vasu ASSISTANT EXAMINER: Kemmerer, Elizabeth C. LEGAL REPRESENTATIVE: Majestic, Parsons, Siebert & Hsue NUMBER OF CLAIMS: EXEMPLARY CLAIM: 1 LINE COUNT: 1061 CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5670481 19970923 . . loss of neurons, whether central, peripheral, or motorneurons. SUMM _ In addition, it may be useful for treating damaged nerve cells, e.g., nerves damaged by traumatic conditions such as burns and wounds, diabetes, kidney dysfunction, and the toxic effects of chemotherapeutics used to treat. sugar alcohols such as mannitol or sorbitol; salt-forming SUMM counterions such as sodium; and/or nonionic surfactants such as Tween, Pluronics or PEG. L17 ANSWER 46 OF 117 USPATFULL Method to enhance permeability of the blood/brain blood/nerve bariers ΤI therapeutic agents ACCESSION NUMBER: 97:86585 USPATFULL TITLE: Method to enhance permeability of the blood/brain blood/nerve bariers to therapeutic agents Poduslo, Joseph F., 5719 St. Mary's Dr. NW, Rochester, MN, United States 55901 INVENTOR(S): Curran, Geoffrey L., 629 23rd St. NE, Rochester, MN, United States 55906 Poduslo, Joseph F., Rochester, MN, United States (U.S. PATENT ASSIGNEE(S): individual) Curran, Geoffrey L., Rochester, MN, United States (U.S.

NUMBER DATE

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PATENT INFORMATION: US 5670477 19970923

individual)

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US 1995-425576 19950420 (8)
APPLICATION INFO.:
                       Utility
DOCUMENT TYPE:
                      MacMillan, Keith
PRIMARY EXAMINER:
LEGAL REPRESENTATIVE: Schwegman, Lundberg, Woessner & Kluth, P.A.
                      21
NUMBER OF CLAIMS:
EXEMPLARY CLAIM:
                       1
                      8 Drawing Figure(s); 8 Drawing Page(s)
NUMBER OF DRAWINGS:
                       1978
LINE COUNT:
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
                                                                 <--
      US 5670477 19970923
                                       . . . cancer (with
DETD
5-fluorouracil);
                               Phase II
              chronic, acute hepatitis B; non-A,
              non-B hepatitis, chronic myelogenous
              leukemia; HIV positive, ARC, AIDS
              (with Retrovir)
Interleukins
PEG-IL-2
            AIDS (with Retrovir)
                               Phase I
Aldesleukin (IL-2)
                               Phase II/III
              Cancer
              Kaposi's sarcoma (with Retrovir)
Human IL-1 alpha
             Bone marrow suppression
     . . . superoxide production. Thus, the delivery of antioxidants,
DETD
such
       as superoxide dismutase, catalase, glutathione peroxide, and the like,
       will limit the neuronal damage caused by free
       radicals in these neurological disorders.
L17 ANSWER 47 OF 117 USPATFULL
    Antibodies to SMDF
                       97:83613 USPATFULL
ACCESSION NUMBER:
TITLE:
                       Antibodies to SMDF
                       Ho, Wei-Hsien, Palo Alto, CA, United States
INVENTOR(S):
                       Osheroff, Phyllis L., Woodside, CA, United States
                       Genentech, Inc., South San Francisco, CA, United
PATENT ASSIGNEE(S):
States
                       (U.S. corporation)
                            NUMBER
                        _____
PATENT INFORMATION: US 5667780 19970916
APPLICATION INFO.: US 1995-428926 19950425 (8)
                                                                   <--
RELATED APPLN. INFO.:
                       Division of Ser. No. US 1994-339517, filed on 14 Nov
                       1994
DOCUMENT TYPE:
                       Utility
                      Feisee, Lila
PRIMARY EXAMINER:
                       Johnson, Nancy A.
ASSISTANT EXAMINER:
LEGAL REPRESENTATIVE: Lee, Wendy M.
                       12
NUMBER OF CLAIMS:
EXEMPLARY CLAIM:
                       1
                      5 Drawing Figure(s); 4 Drawing Page(s)
NUMBER OF DRAWINGS:
                       3743
LINE COUNT:
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       US 5667780 19970916
       SMDF is also believed to find therapeutic use for treating peripheral
     nerve damage (e.g. giant axonal neuropathy, hereditary
```

sensory hypertrophic neuropathy, and sensory neuropathy), leprous neuropathy, Landry-Guillain Barr syndrome, and neuropathy caused by.

DRWD . . . such as mannitol or sorbitol; salt-forming counterions such as sodium; and/or nonionic surfactants such as Tween, Pluronics, or polyethylene glycol (PEG).

DRWD . . . damaged spinal cord in an effort to influence regeneration of interrupted central axons, for assisting in the repair of peripheral nerve injuries and as alternatives to multiple

autografts. See Levi et al., (1994), supra. The use of cell culture techniques to obtain. . .

L17 ANSWER 48 OF 117 USPATFULL

TI Cysteine protease and serine protease inhibitors

ACCESSION NUMBER: 97:73613 USPATFULL

TITLE: Cysteine protease and serine protease inhibitors INVENTOR(S): Mallamo, John P., Glenmore, PA, United States Bihovsky, Ron, Wynnewood, PA, United States

Chatterjee, Sankar, Wynnewood, PA, United States Tripathy, Rabindranath, Pennsville, NJ, United States

PATENT ASSIGNEE(S): Cephalon, Inc., West Chester, PA, United States (U.S.

corporation)

NUMBER DATE

PATENT INFORMATION: US 5658906 19970819 <--

APPLICATION INFO.: US 1996-592074 19960126 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1994-334249, filed on 4 Nov

1994, now patented, Pat. No. US 5498616

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Raymond, Richard L.

LEGAL REPRESENTATIVE: Woodcock Washburn Kurtz Mackiewicz & Norris LLP

NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
LINE COUNT: 1288

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 5658906 19970819

SUMM . . . calpain family of cysteine proteases has been implicated in many diseases and disorders, including neurodegeneration, stroke, Alzheimer's disease, amyotrophy, motor neuron damage, acute central nervous system injury, muscular dystrophy, bone

, acute central nervous system injury, muscular dystrophy, bone resorption, platelet aggregation, cataracts and inflammation. Calpain I has been implicated in. . .

SUMM . . . Sciences (Mack Pub. Co., Easton, Pa., 1980). Formulations for parenteral administration may contain as common excipients sterile water

or saline, **polyalkylene glycols** such as polyethylene glycol, oils of vegetable origin, hydrogenated naphthalenes and the like. In particular, biocompatible, biodegradable lactide polymer lactide/glycolide. . .

L17 ANSWER 49 OF 117 USPATFULL

TI Method and compounds for aica riboside delivery and for lowering blood glucose

ACCESSION NUMBER: 97:73596 USPATFULL

TITLE: Method and compounds for aica riboside delivery and

for

lowering blood glucose

INVENTOR(S): Gruber, Harry E., San Diego, CA, United States
Tuttle, Ronald R., Escondido, CA, United States
Browne, Clinton E., Oceanside, CA, United States

Ugarkar, Bheemarao G., Escondido, CA, United States

Reich, Jack W., Carlsbad, CA, United States Metzner, Ernest K., Del Mar, CA, United States Marangos, Paul J., Encinitas, CA, United States Gensia Pharmaceuticals, Inc., San Diego, CA, United

PATENT ASSIGNEE(S): States (U.S. corporation)

DATE NUMBER

US 5658889 19970819 US 1994-355836 19941214 (8) PATENT INFORMATION: APPLICATION INFO.:

RELATED APPLN. INFO.: Continuation of Ser. No. US 1994-230421, filed on 19

Apr 1994, now abandoned which is a continuation of

Ser.

No. US 1990-466979, filed on 18 Jan 1990, now

abandoned

which is a continuation-in-part of Ser. No. US 1989-301453, filed on 24 Jan 1989, now patented, Pat. No. US 5200525 And Ser. No. US 1989-408107, filed on

15

Sep 1989, now abandoned which is a

continuation-in-part

of Ser. No. US 1989-301222, filed on 24 Jan 1989, now

patented, Pat. No. US 5082829

DOCUMENT TYPE: Utility PRIMARY EXAMINER: Wilson, J

Lyon & Lyon LLP LEGAL REPRESENTATIVE:

NUMBER OF CLAIMS: 22 EXEMPLARY CLAIM:

NUMBER OF DRAWINGS: 34 Drawing Figure(s); 16 Drawing Page(s)

LINE COUNT: 2305

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PΙ US 5658889 19970819

. . . ischemia induced overproduction of the EAA neurotransmitters. DETD Thrombolytic therapy of stroke is therefore not sufficient to protect

against the ensuing neurologic damage after the

occlusion is removed.

. . riboside or one of two prodrugs, compounds 10 and 17 of Table DETD I. The compounds were administered in solution in PEG

400:water (1:1). Results are shown in FIG. 18. A different prodrug. Compound 22 of Table I, was administered in solid. . .

L17 ANSWER 50 OF 117 USPATFULL

Amino acid derivative anticonvulsant ACCESSION NUMBER: 97:68469 USPATFULL

TITLE: Amino acid derivative anticonvulsant

Kohn, Harold L., Houston, TX, United States Watson, Darrell, Belton, TX, United States INVENTOR(S):

Research Corporation Technologies, Inc., Tucson, AZ, PATENT ASSIGNEE(S):

United States (U.S. corporation)

NUMBER DATE _____

US 5654301 19970805 US 1993-3208 19930112 (8) PATENT INFORMATION:

APPLICATION INFO.:

Continuation-in-part of Ser. No. US 1991-710610, filed RELATED APPLN. INFO.: on 4 Jun 1991, now patented, Pat. No. US 5378729 which is a continuation-in-part of Ser. No. US 1989-354057,

filed on 19 May 1989, now abandoned And a

continuation-in-part of Ser. No. US 1989-392870, filed

on 11 Aug 1989, now abandoned , said Ser. No. US

-354057 which is a continuation-in-part of Ser. No. US 1987-80528, filed on 31 Jul 1987, now abandoned which is a continuation-in-part of Ser. No. US 1986-916254, filed on 7 Oct 1986, now abandoned which is a continuation-in-part of Ser. No. US 1985-702195, filed on 15 Feb 1985, now abandoned, said Ser. No. US -392870 which is a continuation of Ser. No. US 1987-80528, filed on 31 Jul 1987, now abandoned which is a continuation-in-part of Ser. No. US 1986-916254, filed on 7 Oct 1986, now abandoned which is a continuation-in-part of Ser. No. US 1985-702195, filed on 15 Feb 1985, now abandoned

NUMBER DATE -----WO 1992-US4687 19920604 PRIORITY INFORMATION: DOCUMENT TYPE: Utility PRIMARY EXAMINER: Criares, Theodore J. LEGAL REPRESENTATIVE: Scully, Scott, Murphy & Presser NUMBER OF CLAIMS: 47 EXEMPLARY CLAIM: 1 LINE COUNT: 4937 CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5654301 19970805 DETD . . . to the top of the screen was determined. Inability to climb to the top within one minute was defined as "neurological impairment". This procedure is described in Pharmacol. Biochem. Behav. 6, 351-353 (1977) and is incorporated herein by reference with the same. . DETD . . in brackets. .sup.b Melting points (.degree.C.) are uncorrected. .sup.c MES = maximal electroshock seizure test. Compound was suspended in 30% **PEG**. .sup.d Tox = neurologic toxicity determined from horizontal screen unless otherwise noted. .sup.e PI = protective index (TD.sub.50 ED.sub.50). f. . DETD . . in brackets. .sup.b Melting points (.degree.C.) are uncorrected. .sup.c MES = maximal electroshock seizure test. Compound was suspended in 30% PEG unless otherwise noted. .sup.d Tox = neurologic toxicity determined from horizontal screen unless otherwise noted. e Not determined. .sup.f Neurologic. . . . >100 DETD >100 ##STR80## >100 ##STR81## >100

L17 ANSWER 51 OF 117 USPATFULL

TI 1,2,3,4-tetrahydroquinoline 2,3,4-trione-3 or 4-oximes

ACCESSION NUMBER: 97:66251 USPATFULL

TITLE: 1,2,3,4-tetrahydroquinoline 2,3,4-trione-3 or 4-oximes

INVENTOR(S): Cai, Sui Xiong, Irvine, CA, United States
Keana, John F. W., Eugene, OR, United States
Weber, Eckard, Laguna Beach, CA, United States

٠.

[.]sup.a MES = maximal electroshock seizure test. Compound was suspended in 30% PEG.

[.]sup.b TOX = neurologic toxicity determined from horizontal screen unless otherwise noted.

The Regents of the University of California, Oakland, PATENT ASSIGNEE(S):

CA, United States (U.S. corporation)

State of Oregon, acting by and through the Oregon

State

Board of Higer Education, acting for and on behalf of the Oregon Health Sciences University, Eugene, OR,

United States (U.S. corporation)

University of Oregon, Eugene, OR, United States (U.S.

corporation)

NUMBER DATE _____

US 5652368 19970729 <--PATENT INFORMATION:

US 1995-536937 19950929 (8) APPLICATION INFO.:

Division of Ser. No. US 1993-69005, filed on 28 May RELATED APPLN. INFO.:

1993, now patented, Pat. No. US 5475007

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Ivy, C. Warren Mach, D. Margaret M. ASSISTANT EXAMINER:

LEGAL REPRESENTATIVE: Sterne, Kessler, Goldstein & Fox P.L.L.C.

NUMBER OF CLAIMS: EXEMPLARY CLAIM: LINE COUNT: 1914

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5652368 19970729

. . from a stroke, the compounds of the present invention may be

administered to ameliorate the immediate ischemia and prevent further

neuronal damage that may occur from recurrent strokes.

SUMM . . . oil, or synthetic fatty acid esters, for example, ethyl oleate or triglycerides or polyethylene glycol-400 (the compounds are soluble in PEG-400). Aqueous injection suspensions may contain

substances which increase the viscosity of the suspension include, for example, sodium carboxymethyl cellulose, sorbitol,. . .

L17 ANSWER 52 OF 117 USPATFULL

Method for treating retinal ganglion cell injury using glial cell line-derived neurothrophic factor (GDNF) protein product

ACCESSION NUMBER:

97:54199 USPATFULL

TITLE:

Method for treating retinal ganglion cell injury using

glial cell line-derived neurothrophic factor (GDNF)

protein product

INVENTOR(S):

Yan, Qiao, Thousand Oaks, CA, United States

Louis, Jean-Claude, Thousand Oaks, CA, United States

PATENT ASSIGNEE(S):

Amgen Inc., Thousand Oaks, CA, United States (U.S.

corporation)

NUMBER DATE

PATENT INFORMATION:

APPLICATION INFO.:

US 5641749 19970624 US 1995-564458 19951129 (8)

DOCUMENT TYPE:

Utility

PRIMARY EXAMINER: Schain, Howard E. ASSISTANT EXAMINER: Touzeau, P. L.

LEGAL REPRESENTATIVE: Curry, Daniel R.; Levy, Ron K.; Odre, Steven M.

NUMBER OF CLAIMS: 20

EXEMPLARY CLAIM: LINE COUNT:

1 1697

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5641749 19970624

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. . protein product increases the in vivo survival of injured DETD retinal ganglion cells, which cells make up the main population of

```
neurons damaged in glaucoma. It is postulated that
       administration of exogenous GDNF protein product will protect retinal
       ganglion cells from traumatic damage.
       Suitable water soluble polymers include, but are not limited to,
DETD
      polyethylene glycol (PEG), copolymers of ethylene
       glycol/propylene glycol, carboxymethylcellulose, dextran, polyvinyl
       alcohol, polyvinyl pyrrolidone, poly-1, 3-dioxolane,
      poly-1,3,6-trioxane, ethylene/maleic anhydride copolymer,
polyaminoacids
       (either homopolymers.
       . . in the art. See for example, EP 0 401 384, the disclosure of
DETD
       which is hereby incorporated by reference (coupling PEG to
       G-CSF), see also Malik et al., Exp. Hematol., 20:1028-1035, 1992
       (reporting pegylation of GM-CSF using tresyl chloride). For example,.
       . . . reacting an active ester derivative of polyethylene glycol
DETD
with
       the GDNF protein or variant. Any known or subsequently discovered
       reactive PEG molecule may be used to carry out the pegylation
       of GDNF protein or variant. A preferred activated PEG ester is
     PEG esterified to N-hydroxysuccinimide. As used herein,
       "acylation" is contemplated to include without limitation the following
       types of linkages between the therapeutic protein and a water soluble
       polymer such as PEG: amide, carbamate, urethane, and the like.
       See Bioconjugate Chem., 5:133-140, 1994. Reaction conditions may be
       selected from any of those. .
       Pegylation by alkylation generally involves reacting a terminal
DETD
aldehyde
       derivative of PEG with the GDNF protein or variant in the
       presence of a reducing agent. Pegylation by alkylation can also result
       in. . . the N-terminus of the GDNF protein or variant (i.e., a
       mono-pegylated protein). In either case of monopegylation or
       polypegylation, the PEG groups are preferably attached to the
      protein via a \operatorname{--CH2-NH--} group. With particular reference to the
--CH2--
       group, this type. . .
       . . . protein products to be used in accordance with the present
DETD
       invention may include pegylated GDNF protein or variants, wherein the
     PEG group(s) is (are) attached via acyl or alkyl groups. As
       discussed above, such products may be mono-pegylated or poly-pegylated
       (e.g., containing 2-6, and preferably 2-5, PEG groups). The
     PEG groups are generally attached to the protein at the a- or
       e-amino groups of amino acids, but it is also contemplated that the
     PEG groups could be attached to any amino group attached to the
       protein, which is sufficiently reactive to become attached to a
     PEG group under suitable reaction conditions.
DETD
       . . . preferably, so that the degree of polymerization may be
       controlled as provided for in the present methods. An exemplary
reactive
     PEG aldehyde is polyethylene glycol propionaldehyde, which is
       water stable, or mono C1-C10 alkoxy or aryloxy derivatives thereof
(see,
             . for use herein is polyethylene glycol. As used herein,
DETD
       polyethylene glycol is meant to encompass any of the forms of
     PEG that have been used to derivatize other proteins, such as
       mono-(C1-C10) alkoxy- or aryloxy-polyethylene glycol.
DETD
            . of (a) reacting a GDNF protein or variant with polyethylene
       glycol (such as a reactive ester or aldehyde derivative of PEG
       ) under conditions whereby the protein becomes attached to one or more
```

PEG groups, and (b) obtaining the reaction product(s). In general, the optimal reaction conditions for the acylation reactions will be determined case-by-case based on known parameters and the desired result. For example, the larger the ratio of PEG :protein, the greater the percentage of poly-pegylated product. . . . (or variant) conjugate molecule will generally comprise the DETD steps of: (a) reacting a GDNF protein or variant with a reactive PEG molecule under reductive alkylation conditions, at a pH suitable to permit selective modification of the a-amino group at the . . . that treatment with GDNF protein product increases the DETD of injured retinal ganglion cells, which are the main population of neurons damaged in glaucoma. L17 ANSWER 53 OF 117 USPATFULL Phosphorous-containing cysteine and serine protease inhibitors ACCESSION NUMBER: 97:51979 USPATFULL TITLE: Phosphorous-containing cysteine and serine protease inhibitors Mallamo, John P., Glenmore, PA, United States INVENTOR(S): Bihovsky, Ron, Wynnewood, PA, United States Tao, Ming, Maple Glen, PA, United States Wells, Gregory J., West Chester, PA, United States Cephalon, Inc., West Chester, PA, United States (U.S. PATENT ASSIGNEE(S): corporation) NUMBER DATE PATENT INFORMATION: US 5639732 19970617 APPLICATION INFO.: US 1996-679342 19960710 (8) <--NUMBER DATE ______ PRIORITY INFORMATION: US 1995-1491 19950717 (60) PRIMARY EXAMINER: Richter, Johann
ASSISTANT EXAMINER: Ambrose, Michael G.
LEGAL REPRESENTATIVE: Woodcock Washburn Kurtz Mackiewicz & Norris LLP
NUMBER OF CLAIMS: 44 DOCUMENT TYPE: Utility NUMBER OF CLAIMS: 44 EXEMPLARY CLAIM: 1 1567 LINE COUNT: CAS INDEXING IS AVAILABLE FOR THIS PATENT. ΡI US 5639732 19970617 SUMM . . . The calpain family of cysteine proteases has been implicated in many diseases and disorders, including neurodegeneration, stroke, Alzheimer's, amyotrophy, motor neuron damage, acute central nervous system injury, muscular dystrophy, bone resorption, platelet aggregation, cataracts and inflammation. Calpain I has been implicated in. Sciences (Mack Pub. Co., Easton, Pa., 1980). Formulations for SUMM parenteral administration may contain as common excipients sterile water or saline, polyalkylene glycols such as polyethylene glycol, oils and vegetable origin, hydrogenated naphthalenes and the

L17 ANSWER 54 OF 117 USPATFULL
TI Alkyl, azido, alkoxy, and fluoro-substituted and fused
quinoxalinediones

lactide/glycolide.

like. In particular, biocompatible, biodegradable lactide polymer,

ACCESSION NUMBER:

97:42998 USPATFULL

TITLE:

Alkyl, azido, alkoxy, and fluoro-substituted and fused

quinoxalinediones

INVENTOR(S):

Cai, Sui X., Irvine, CA, United States

Weber, Eckard, Laguna Beach, CA, United States Keana, John F.W., Eugene, OR, United States

Kher, Sunil, Eugene, OR, United States

PATENT ASSIGNEE(S):

State of Oregon, acting by and through the Oregon

State

Board of Higher Education, acting for and on behalf of

the Oregon Health Sciences University and the University of Oregon, Eugene Oregon, Eugene, OR,

United

States (U.S. corporation)

Acea Pharmaceuticals, Inc., Irvine, CA, United States

(U.S. corporation)

The Regents of the University of California, Oakland,

CA, United States (U.S. corporation)

NUMBER DATE

PATENT INFORMATION:

US 5631373 19970520

APPLICATION INFO.:

US 1994-289603 19940811 (8)

RELATED APPLN. INFO.:

Continuation-in-part of Ser. No. US 1994-208878, filed

<---

on 11 Mar 1994, now abandoned which is a

continuation-in-part of Ser. No. US 1993-148268, filed

on 5 Nov 1993, now abandoned And Ser. No. US

1993-148259, filed on 5 Nov 1993, now patented, Pat.

No. US 5514680

DOCUMENT TYPE:

Utility

PRIMARY EXAMINER:

Burn, Brian M.

LEGAL REPRESENTATIVE: St

Sterne, Kessler, Goldstein & Fox P.L.L.C.

NUMBER OF CLAIMS:

10

EXEMPLARY CLAIM: NUMBER OF DRAWINGS:

10 Drawing Figure(s); 9 Drawing Page(s)

LINE COUNT:

4381

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 5631373 19970520

20

 ${\tt DETD}$. . been diagnosed as suffering from a stroke, the compounds can be

administered to ameliorate the immediate ischemia and prevent further neuronal damage that may occur from recurrent strokes.

DETD . . . oil, or synthetic fatty acid esters, for example, ethyl oleate or triglycerides or polyethylene glycol-400 (the compounds are soluble in PEG-400). Aqueous injection suspensions may contain substances that increase the viscosity of the suspension, for example, sodium carboxymethyl cellulose, sorbitol, and/or. . .

L17 ANSWER 55 OF 117 USPATFULL

TI 4-hydroxy-3-nitro-1,2-dihydroquinolin-2-ones and the use thereof as

excitatory amino acid and glycine receptor antagonists

ACCESSION NUMBER:

97:33760 USPATFULL

TITLE:

4-hydroxy-3-nitro-1,2-dihydroquinolin-2-ones and the

use thereof as excitatory amino acid and glycine

receptor antagonists

INVENTOR(S):

Cai, Sui X., Irvine, CA, United States

Weber, Eckard, Laguna Beach, CA, United States Keana, John F. W., Eugene, OR, United States

PATENT ASSIGNEE(S):

State of Oregon, acting by and through The Oregon

State

Board of Higher Education, acting for and on behalf of

The Oregon Health Sciences University and The University of Oregon, Eugene Oregon, Eugene, OR,

United

States (U.S. corporation)

The Regents of the University of California, Oakland,

CA, United States (U.S. corporation)

DATE NUMBER _____

US 5622965 19970422 PATENT INFORMATION: <--

APPLICATION INFO.: US 1993-101244 19930802 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1993-30608, filed

on 12 Mar 1993, now abandoned

DOCUMENT TYPE: Utility

Ivy, C. Warren PRIMARY EXAMINER: Mach, D. Margaret M. ASSISTANT EXAMINER:

LEGAL REPRESENTATIVE: Sterne, Kessler, Goldstein & Fox P.L.L.C.

NUMBER OF CLAIMS: EXEMPLARY CLAIM: LINE COUNT: 1507

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5622965 19970422

SUMM . . . from a stroke, the compounds of the present invention may be administered to ameliorate the immediate ischemia and prevent further

neuronal damage that may occur from recurrent strokes.

. . . oil, or synthetic fatty acid esters, for example, ethyl oleate or triglycerides or polyethylene glycol-400 (the compounds are soluble in PEG-400). Aqueous injection suspensions may contain

substances which increase the viscosity of the suspension include, for example, sodium carboxymethyl cellulose, sorbitol,. . .

L17 ANSWER 56 OF 117 USPATFULL

Glycine receptor antagonists and the use thereof

ACCESSION NUMBER: 97:33747 USPATFULL

TITLE: Glycine receptor antagonists and the use thereof INVENTOR(S):

Weber, Eckard, Laguna Beach, CA, United States Keana, John F. W., Eugene, OR, United States

PATENT ASSIGNEE(S): State of Oregon, acting by and through the Oregon

State

Board of Higher Education, acting for and on behalf of

the Oregon Health Sciences University and the University of Oregon, Eugene Oregon, Eugene, OR,

United

States (U.S. corporation)

The Regents of the University of California, Oakland,

CA, United States (U.S. corporation)

NUMBER DATE

US 5622952 19970422 PATENT INFORMATION: APPLICATION INFO.: US 1995-405713 19950317 (8)

Division of Ser. No. US 1993-148259, filed on 5 Nov RELATED APPLN. INFO.:

1993, now patented, Pat. No. US 5514680 which is a continuation-in-part of Ser. No. US 1993-69274, filed

on 28 May 1993, now abandoned which is a

continuation-in-part of Ser. No. US 1992-995167, filed

on 22 Dec 1992, now abandoned which is a

continuation-in-part of Ser. No. US 1992-903080, filed

on 22 Jun 1992, now abandoned

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Dees, Jos e G.

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ASSISTANT EXAMINER:
                       Cebulak, Mary C.
LEGAL REPRESENTATIVE:
                       Sterne, Kessler, Goldstein & Fox P.L.L.C.
NUMBER OF CLAIMS:
                        19
EXEMPLARY CLAIM:
                        1
NUMBER OF DRAWINGS:
                        45 Drawing Figure(s); 45 Drawing Page(s)
LINE COUNT:
                        7443
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       US 5622952 19970422
         . . from a stroke, the compounds of the present invention may be
       administered to ameliorate the immediate ischemia and prevent further
    neuronal damage that may occur from recurrent strokes.
       . . . oil, or synthetic fatty acid esters, for example, ethyl oleate
       or triglycerides or polyethylene glycol-400 (the compounds are soluble
       in PEG-400). Aqueous injection suspensions may contain
       substances which increase the viscosity of the suspension include, for
       example, sodium carboxymethyl cellulose, sorbitol,. . .
DETD
      Also conducted were dose-response experiments i.v. using a
TRIS/TWEE-80/
     PEG-400 formulation as a vehicle for the drug. The ED.sub.50
       value (5-6 mg/kg) for 5-NO.sub.2 -6,7-Cl.sub.2 -QX in this formulation
DETD
       . . . extended to both locomotor activity and radial arm maze
       performance. Consistent with this robust behavioral protection,
       5-chloro-7-trifluoromethyl-1,4-dihydroquinoxaline-2,3-dione also
      protected against neuronal damage.
      A 5 mg/ml solution of 5-nitro-6,7-dichloro-1,4-dihydroquinoxaline-2,3-
      dione was prepared by dissolving the 5-nitro-6,7-quinoxaline-2,3-dione
       in an aqueous solution containing 10% polyethyleneglycol 400 (
     PEG-400), 0.45% TWEEN-80 and 0.18M TRIS (Tromethamine) to give a
       final concentration of 5 mg/ml of 5-nitro-6,7-dichloro-1,4-
       dihydroquinoxaline-2,3-dione. The 5-nitro-6,7-dichloro-1,4-
      dihydroquinoxaline-2,3-dione readily dissolved. . . least 1-2 years.
      A 10 mg/ml solution of 5-nitro-6,7-dichloro-1,4-dihydroquinoxaline-2,3-
      dione was prepared by dissolving the compound in a solution containing
       50% PEG-400, 0.5% TWEEN-80 and 0.1M TRIS (Tromethamine). The
       5-nitro-6,7-dichloro-1,4-dihydroquinoxalineo2,3-dione readily dissolved
       in this solution by warming to 60.degree.-100.degree. C. The solution.
         . this solution will be stable for at least \bar{1}-2 years. A 5 mg/ml
       solution of 5-nitro-6,7-dichloro-1,4-dihydroquinoxaline-2,3-dione was
       also prepared without PEG-400 by dissolving the compound in a
       solution containing 0.05M TRIS (Tromethamine), 0.5% Tween-80 and 5%
       glucose. The solution was sterilized.
      A 10 mg/ml solution of
5-chloro-7-trifluoromethyl-1, 4-dihydroquinoxaline-
       2,3-dione was prepared by dissolving the compound in 0.1M
      bis-tris-propane, 50% PEG-400 or propyleneglycol, 0.75%
       TWEEN-80. The compound dissolved readily by warming in a boiling water
      bath. The solution was autoclaved and. .
L17 ANSWER 57 OF 117 USPATFULL
       Protein kinase inhibitors for treatment of neurological disorders
ACCESSION NUMBER:
                        97:31819 USPATFULL
TITLE:
                        Protein kinase inhibitors for treatment of
neurological
                        disorders
INVENTOR(S):
                        Lewis, Michael E., West Chester, PA, United States
                        Kauer, James C., Kennett Square, PA, United States
                        Neff, Nicola, Wallingford, PA, United States
                        Roberts-Lewis, Jill, West Chester, PA, United States
                        Murakata, Chikara, Hachioji, Japan
```

Saito, Hiromitsu, Mishima, Japan

Matsuda, Yuzuru, Koganei, Japan

Glicksman, Marcie A., Swarthmore, PA, United States

Kanai, Fumihiko, Machida, Japan Kaneko, Masami, Sagamihara, Japan

PATENT ASSIGNEE(S): Cephalon, Inc., West Chester, PA, United States (U.S.

corporation)

Kyowa Hakko Kogyo, Tokyo, Japan (non-U.S. corporation)

NUMBER DATE

PATENT INFORMATION:

US 5621101 19970415

APPLICATION INFO.:

US 1995-486739 19950607 (8)

RELATED APPLN. INFO .:

Continuation-in-part of Ser. No. US 1994-329540, filed

on 26 Oct 1994, now patented, Pat. No. US 5621100

which

is a continuation-in-part of Ser. No. US 1993-96561,

filed on 22 Jul 1993, now patented, Pat. No. US

5461146

which is a continuation-in-part of Ser. No. US 1992-920102, filed on 24 Jul 1992, now abandoned

DOCUMENT TYPE:

PRIMARY EXAMINER:

Utility Shah, Mukund J.

ASSISTANT EXAMINER:

Sripada, Pavanaram K. Fish & Richardson P.C.

LEGAL REPRESENTATIVE: NUMBER OF CLAIMS:

16

EXEMPLARY CLAIM: NUMBER OF DRAWINGS:

27 Drawing Figure(s); 22 Drawing Page(s)

LINE COUNT:

2840 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5621101 19970415

. . . Sciences (Mack Pub. Co, Easton, Pa., 1980). Formulations for parenteral administration may contain as common excipients sterile

water

or saline, polyalkylene glycols such as polyethylene glycol, oils of vegetable origin, hydrogenated naphthalenes and the like. In particular, biocompatable, biodegradable lactide polymer, lactide/glycolide. . .

DETD Kainate infusion regime: The effect of K-252a or its derivatives on kainate-induced neuronal damage was evaluated. Adult male or female Sprague-Dawley rats (175-250 g) were anesthetized with Nembutal (50 mg/kg, ip). Each rat was. .

L17 ANSWER 58 OF 117 USPATFULL

K-252a derivatives for treatment of neurological disorders

ACCESSION NUMBER:

97:31818 USPATFULL

TITLE:

K-252a derivatives for treatment of neurological

disorders

INVENTOR(S):

Lewis, Michael E., West Chester, PA, United States Kauer, James C., Kennett Square, PA, United States

Neff, Nicola, Wallingford, PA, United States

Roberts-Lewis, Jill, West Chester, PA, United States

Murakata, Chikara, Hachioji, Japan Saito, Hiromitsu, Mishima, Japan Matsuda, Yuzuru, Koganei, Japan

Glicksman, Marcie A., Swarthmore, PA, United States

Kanai, Fumihiko, Machida, Japan Kaneko, Masami, Sagamihara, Japan

PATENT ASSIGNEE(S):

Cephalon, Inc., West Chester, PA, United States (U.S.

corporation)

Kyowa Hakko Kogyo Co., Ltd., Tokyo, Japan (non-U.S.

corporation)

DATE NUMBER ______ PATENT INFORMATION: US 5621100 19970415 US 1994-329540 19941026 (8) <--APPLICATION INFO.: RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1993-96561, filed on 22 Jul 1993, now patented, Pat. No. US 5461146 which is a continuation-in-part of Ser. No. US 1992-920102, filed on 24 Jul 1992, now abandoned DOCUMENT TYPE: Utility PRIMARY EXAMINER: Shah, Mukund J. ASSISTANT EXAMINER: Sripada, Pavanaram K. LEGAL REPRESENTATIVE: Fish & Richardson P.C. NUMBER OF CLAIMS: EXEMPLARY CLAIM: NUMBER OF DRAWINGS: 26 Drawing Figure(s); 20 Drawing Page(s) LINE COUNT: 2356 CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5621100 19970415 DETD . . . Sciences (Mack Pub. Co, Easton, Pa., 1980). Formulations for parenteral administration may contain as common excipients sterile water or saline, polyalkylene glycols such as polyethylene glycol, oils of vegetable origin, hydrogenated naphthalenes and the like. In particular, biocompatable, biodegradable lactide polymer, lactide/glycolide. . . DETD Kainate infusion regime: The effect of K-252a or its derivatives on kainate-induced neuronal damage was evaluated. Adult male or female Sprague-Dawley rats (175-250 g) were anesthetized with Nembutal (50 mg/kg, ip). Each rat was. . . L17 ANSWER 59 OF 117 USPATFULL Glial mitogenic factors ACCESSION NUMBER: 97:31800 USPATFULL TITLE: Glial mitogenic factors INVENTOR(S): Goodearl, Andrew, Chorleywood, United Kingdom Stroobant, Paul, London, United Kingdom Minghetti, Luisa, Bagnacavallo, Italy Waterfield, Michael, Newbury, United Kingdom Marchioni, Mark, Arlington, MA, United States Chen, Mario S., Arlington, MA, United States Hiles, Ian, London, England Ludwig Institute for Cancer Research, NY, United PATENT ASSIGNEE(S): States (U.S. corporation) Cambridge Neuroscience, Cambridge, MA, United States (U.S. corporation) NUMBER DATE -----US 5621081 19970415 US 1995-471855 19950606 (8) PATENT INFORMATION: <--APPLICATION INFO.: Division of Ser. No. US 1993-36555, filed on 24 Mar RELATED APPLN. INFO.: 1993 which is a continuation-in-part of Ser. No. US 1992-863703, filed on 3 Apr 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-907138, filed on 30 Jun 1992, now abandoned which is a

continuation-in-part of Ser. No. US 1992-940389, filed

continuation-in-part of Ser. No. US 1992-965173, filed

on 3 Sep 1992, now abandoned which is a

```
on 23 Oct 1992, now abandoned
DOCUMENT TYPE:
                         Utility
 PRIMARY EXAMINER:
                         Walsh, Stephen G. Gucker, Stephen
ASSISTANT EXAMINER:
LEGAL REPRESENTATIVE:
                         Felfe & Lynch; Butler, Gregory B.
NUMBER OF CLAIMS:
EXEMPLARY CLAIM:
NUMBER OF DRAWINGS:
                         89 Drawing Figure(s); 78 Drawing Page(s)
LINE COUNT:
                         3290
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       US 5621081 19970415
SUMM
       Included in the invention as well, are methods for treatment when the
       condition involves peripheral nerve damage;
     nerve damage in the central nervous system;
       neurodegenerative disorders; demyelination in peripheral or central
       nervous system; or damage or loss of Schwann. . .
SUMM
          . . in, for example, "Remington's Pharmaceutical Sciences."
       Formulations for parenteral administration may, for example, contain as
       excipients sterile water or saline, polyalkylene
     glycols such as polyethylene glycol, oils of vegetable origin,
       or hydrogenated naphthalenes, biocompatible, biodegradable lactide
       polymer, or polyoxyethylene-polyoxypropylene copolymers may be. . .
L17 ANSWER 60 OF 117 USPATFULL
       Glycine receptor antagonists and the use thereof
ACCESSION NUMBER:
                         97:31701 USPATFULL
TITLE:
                         Glycine receptor antagonists and the use thereof
INVENTOR(S):
                         Weber, Eckard, Laguna Beach, CA, United States
                         Keana, John F. W., Eugene, OR, United States
PATENT ASSIGNEE(S):
                         State of Oregon, Acting By and Through The Oregon
State
                         Board of Higher Education, Acting For and On Behalf of
                         The Oregon Health Sciences University and The
                         University of Oregon, Eugene Oregon, Eugene, OR,
United
                         States (U.S. corporation)
                         The Regents of The University of California, Oakland,
                         CA, United States (U.S. corporation)
                              NUMBER
                                         DATE
                         US 5620979 19970415
PATENT INFORMATION:
                                                                      <--
                         US 1995-405708 19950317 (8)
APPLICATION INFO.:
                         Division of Ser. No. US 1993-148259, filed on 5 Nov
RELATED APPLN. INFO.:
                         1993, now patented, Pat. No. US 5514680 which is a continuation-in-part of Ser. No. US 1993-69274, filed
                         on 28 May 1993, now abandoned which is a
                         continuation-in-part of Ser. No. US 1992-995167, filed
                         on 22 Dec 1992, now abandoned which is a
                         continuation-in-part of Ser. No. US 1992-903080, filed
                         on 22 Jun 1992, now abandoned
DOCUMENT TYPE:
                        Utility
PRIMARY EXAMINER:
                        Dees, Jose G.
ASSISTANT EXAMINER:
                        Cebulak, Mary C.
LEGAL REPRESENTATIVE:
                        Sterne, Kessler, Goldstein & Fox P.L.L.C.
NUMBER OF CLAIMS:
EXEMPLARY CLAIM:
NUMBER OF DRAWINGS:
                        45 Drawing Figure(s); 45 Drawing Page(s)
LINE COUNT:
                        7507
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       US 5620979 19970415
                                                                      <--
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. from a stroke, the compounds of the present invention may be
                  administered to ameliorate the immediate ischemia and prevent further
             neuronal damage that may occur from recurrent strokes.
                         . . oil, or synthetic fatty acid esters, for example, ethyl oleate
                  or triglycerides or polyethylene glycol-400 (the compounds are soluble
                  in PEG-400). Aqueous injection suspensions may contain
                  substances which increase the viscosity of the suspension include, for
                  example, sodium carboxymethyl cellulose, sorbitol,. .
                  Also conducted were dose-response experiments i.v. using a
 DETD
                  TRIS/TWEEN-80/PEG-400 formulation as a vehicle for the drug.
                  The ED.sub.50 value (5-6 \text{ mg/kg}) for 5-\text{NO.sub.2} -6,7-\text{Cl.sub.2} -\text{QX} in
 this
                  formulation was.
 DETD
                  . . . extended to both locomotor activity and radial arm maze % \left( 1\right) =\left( 1\right) +\left( 1
                 performance. Consistent with this robust behavioral protection,
                  5-chloro-7-trifluoromethyl-1,4-dihydroquinoxaline-2,3-dione also
                 protected against neuronal damage.
 DETD
                         . . 5 mg/ml solution of
 5-nitro-6,7-dichloro-1,4-dihydroquinoxaline-
                  2,3-dione was prepared by dissolving the 5-nitro-6,7-quinoxaline-2,3-
                 dione in an aqueous solution containing 10 9 polyethyleneglycol 400 (
             PEG-400), 0.45% TWEEN 80 and 0.18M TRIS (Tromethamine) to give a
                 final concentration of 5 mg/ml of 5-nitro-6,7-dichloro-1,4-
                 dihydroquinoxaline-2,3-dione. The 5-nitro-6,7-dichloro-1,4-
                 dihydroquinoxaline-2,3-dione readily. . . least 1-2 years. A 10
mg/ml
                 solution of 5-nitro-6,7-dichloro-1,4-dihydroquinoxaline-2,3-dione was
                 prepared by dissolving the compound in a solution containing 50%
            PEG-400, 0.5% TWEEN-80 and 0.1M Tris (Tromethamine). The
                 5-nitro-6,7-dichloro-1,4-dihydroquinoxaline-2,3-dione readily dissolved
                 in this solution by warming to 60.degree.-100.degree. C. The solution.
                        . this solution will be stable for at least 1-2 years. A 5 mg/ml
                 solution of 5-nitro-6,7-dichloro-1,4-dihydroquinoxaline-2,3-dione was
                 also prepared without PEG-400 by dissolving the compound in a
                 solution containing 0.05M TRIS (Tromethamine), 0.59.TWEEN-80 and 5%
                 glucose. The solution was sterilized and. .
                 A 10 mg/ml solution of
5-chloro-7-trifluoromethyl-1,4-dihydroquinoxaline-
                 2,3-dione was prepared by dissolving the compound in 0.1M
                bis-tris-propane, 50% PEG-400 or propyleneglycol, 0.75%
                 TWEEN-80. The compound dissolved readily by warming in a boiling water
                bath. The solution was autoclaved and. .
L17 ANSWER 61 OF 117 USPATFULL
ΤI
                8-aza, 6-aza and 6,8-diaza-1,4-dihydroquinoxaline-2,3-diones and the
use
                thereof as antagonists for the glycine/NMDA receptor
ACCESSION NUMBER:
                                                         97:31700 USPATFULL
TITLE:
                                                         8-aza, 6-aza and 6,8-diaza-1,4-dihydroquinoxaline-2,3-
                                                         diones and the use thereof as antagonists for the
                                                         glycine/NMDA receptor
INVENTOR(S):
                                                         Cai, Sui X., Irvine, CA, United States
                                                         Keana, John F. W., Eugene, OR, United States
                                                         Weber, Eckard, Laguna Beach, CA, United States
PATENT ASSIGNEE(S):
                                                         State of Oregon, acting by and through The Oregon
State
                                                         Board of Higher Education, acting for and on behalf of
                                                         The Oregon Health Sciences University and The
                                                         University of Oregon, Eugene Oregon, Eugene, OR,
United
                                                         States (U.S. corporation)
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The Regents of the University of California, Oakland, CA, United States (U.S. corporation) ACEA Pharmaceuticals, Inc., Irvine, CA, United States (U.S. corporation)

DATE NUMBER -----

PATENT INFORMATION: US 5620978 19970415 US 1995-368163 19950103 (8) APPLICATION INFO.:

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1994-289366, filed

on 11 Aug 1994, now abandoned which is a

continuation-in-part of Ser. No. US 1994-176278, filed

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<--

on 3 Jan 1994, now abandoned

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Shah, Mukund J. ASSISTANT EXAMINER: Grumbling, Matthew V.

LEGAL REPRESENTATIVE: Sterne, Kessler, Goldstein & Fox P.L.L.C.

NUMBER OF CLAIMS: EXEMPLARY CLAIM:

NUMBER OF DRAWINGS: 7 Drawing Figure(s); 5 Drawing Page(s)

LINE COUNT: 2779

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5620978 19970415

DETD . . . from a stroke, the compounds of the present invention may be administered to ameliorate the immediate ischemia and prevent further

neuronal damage that may occur from recurrent strokes.

. . . oil, or synthetic fatty acid esters, for example, ethyl oleate or triglycerides or polyethylene glycol-400 (the compounds are soluble in PEG-400). Aqueous injection suspensions can contain substances that increase the viscosity of the suspension, for example, sodium carboxymethyl cellulose, sorbitol, and/or. .

L17 ANSWER 62 OF 117 USPATFULL

Process for preparing glial mitogenic factors

ACCESSION NUMBER: 97:16183 USPATFULL

TITLE: Process for preparing glial mitogenic factors INVENTOR(S): Goodearl, Andrew, Hertfordshire, United Kingdom

> Stroobant, Paul, London, United Kingdom Minghetti, Luisa, Bagnacavallo, Italy

Waterfield, Michael, Berkshire, United Kingdom Marchioni, Mark, Arlington, MA, United States Chen, Mario S., Arlington, MA, United States

Hiles, Ian, London, England

PATENT ASSIGNEE(S): Ludwig Institute for Cancer Research, New York, NY,

United States (U.S. corporation)

Cambridge Neuroscience, Cambridge, MA, United States

(U.S. corporation)

NUMBER DATE

PATENT INFORMATION: APPLICATION INFO.:

US 5606032 19970225 US 1995-469569 19950606 (8)

RELATED APPLN. INFO.:

Division of Ser. No. US 1993-36555, filed on 24 Mar 1993 which is a continuation-in-part of Ser. No. US 1992-863703, filed on 3 Apr 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-907138,

filed on 30 Jun 1992, now abandoned which is a

continuation-in-part of Ser. No. US 1992-940389, filed

on 3 Sep 1992, now abandoned which is a

continuation-in-part of Ser. No. US 1992-965173, filed

on 23 Oct 1992, now abandoned

NUMBER DATE -----PRIORITY INFORMATION: GB 1991-7566 19910410 DOCUMENT TYPE: Utility Schain, Howard F. PRIMARY EXAMINER: LEGAL REPRESENTATIVE: Felfe & Lynch; Butler, Gregory B. NUMBER OF CLAIMS: 12 EXEMPLARY CLAIM: NUMBER OF DRAWINGS: 88 Drawing Figure(s); 78 Drawing Page(s) LINE COUNT: 3346 CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5606032 19970225 SUMM Included in the invention as well, are methods for treatment when the condition involves peripheral nerve damage; nerve damage in the central nervous system; neurodegenerative disorders; demyelination in peripheral or central nervous system; or damage or loss of Schwann. . . in, for example, "Remington's Pharmaceutical Sciences." SUMM Formulations for parenteral administration may, for example, contain as excipients sterile water or saline, polyalkylene glycols such as polyethylene glycol, oils of vegetable origin, or hydrogenated naphthalenes, biocompatible, biodegradable lactide polymer, or polyoxyethylene-polyoxypropylene copolymers may be. . . L17 ANSWER 63 OF 117 USPATFULL Production and purification of biologically active recombinant neurotrophic protein in bacteria ACCESSION NUMBER: 97:16182 USPATFULL TITLE: Production and purification of biologically active recombinant neurotrophic protein in bacteria INVENTOR(S): Lile, Jack, 947 Casitas Vista Rd., Ventura, CA, United States 93001 Kohno, Tadahiko, 1557 Hays Ct., Louisville, CO, United States 80027 Bonam, Duane, 4 Morsecroft La., Amesbury, MA, United States 01913 Rosendahl, Mary S., 310 Fairplay, Broomsfield, CO, United States 80020 NUMBER DATE PATENT INFORMATION: US 5606031 19970225 <--APPLICATION INFO.: US 1994-266080 19940627 (8) RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1994-240122, filed on 9 May 1994, now abandoned which is a continuation-in-part of Ser. No. US 1993-87912, filed on 6 Jul 1993, now abandoned which is a continuation of Ser. No. US 1991-680681, filed on 4 Apr 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-594126, filed on 9 Oct 1990, now patented, Pat.

No. US 5235043 Ser. No. Ser. No. US 1990-547750, filed on 2 Jul 1990, now abandoned And Ser. No. US

1990-505441, filed on 6 Apr 1990, now abandoned

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Allen, Marianne P.

LEGAL REPRESENTATIVE: Swanson & Bratschun LLC

NUMBER OF CLAIMS: 22 EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 13 Drawing Figure(s); 12 Drawing Page(s) LINE COUNT: 1136 CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5606031 19970225 In order for a particular neurotrophic factor to be potentially useful SUMM in treating nerve damage, it must be available in sufficient quantity to be used as a pharmaceutical treatment. Also, since neurotrophic factors are proteins,. SUMM Another method to improve the recovery of biologically active protein from bacterial expression systems includes the use of polyethylene glycol (PEG) in the refolding mixture. It has been proposed that the addition of PEG prevents protein aggregation resulting from the association of hydrophobic intermediates in the refolding pathway. Cleland et al. (1990) Biotechnology 8:1274. (1992) J. Biol. Chem. 267:13327, reported improved recovery of biologically active bovine carbonic anhydrase B (CAB) with the addition of PEG during the refolding process. The concentration of PEG required to achieve an increase in the recovery of active protein was twice the total protein concentration, and required PEG with molecular weights of 1000-8000 (Cleland et al. (1992) supra). SUMM then oxidized, and the protein allowed to form the correct disulfide bonds. The refolding mixture preferably contained up to 25%**PEG** 200 or 300. Sulfonylated neurotrophic factor is purified by anion exchange SUMM chromatography and refolded in the presence of 20% polyethylene glycol 300 (PEG 300). Refolded neurotrophic factor is purified by cation exchange chromatógraphy. L17 ANSWER 64 OF 117 USPATFULL Method of using a secretable glial mitogenic factor to induce acetylcholine receptor synthesis ACCESSION NUMBER: 97:12435 USPATFULL TITLE: Method of using a secretable glial mitogenic factor to induce acetylcholine receptor synthesis INVENTOR(S): Goodearl, Andrew, Chorleywood, United Kingdom Stroobant, Paul, London, United Kingdom Minghetti, Luisa, Bagnacavallo, Italy Waterfield, Michael, Newbury, United Kingdom Marchioni, Mark, Arlington, MA, United States Chen, Mario S., Arlington, MA, United States Hiles, Ian, London, England PATENT ASSIGNEE(S): Ludwig Institute for Cancer Research, New York, NY, United States (U.S. corporation) Cambridge Neuroscience Research Inc., Cambridge, MA, United States (U.S. corporation) NUMBER -----PATENT INFORMATION: US 5602096 19970211 <--US 1995-472008 19950606 (8) APPLICATION INFO.: Division of Ser. No. US 1993-36555, filed on 24 Mar RELATED APPLN. INFO.: 1993, now patented, Pat. No. US 5530109 which is a continuation-in-part of Ser. No. US 1992-965173, filed

NUMBER DATE

1992, now abandoned

on 23 Oct 1992, now abandoned Ser. No. Ser. No. US 1992-940389, filed on 3 Sep 1992, now abandoned Ser. No. Ser. No. US 1992-907138, filed on 30 Jun 1992, now abandoned And Ser. No. US 1992-863703, filed on 3 Apr

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PRIORITY INFORMATION: GB 1991-7566 19910410
DOCUMENT TYPE:
                        Utility
PRIMARY EXAMINER:
                       Walsh, Stephen G.
ASSISTANT EXAMINER:
                       Gucker, Stephen
LEGAL REPRESENTATIVE: Felfe & Lynch; Butler, Gregory B.
NUMBER OF CLAIMS:
EXEMPLARY CLAIM:
NUMBER OF DRAWINGS:
                       89 Drawing Figure(s); 78 Drawing Page(s)
LINE COUNT:
                        3304
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       US 5602096 19970211
SUMM
       Included in the invention as well, are methods for treatment when the
       condition involves peripheral nerve damage;
     nerve damage in the central nervous system;
       neurodegenerative disorders; demyelination in peripheral or central
       nervous system; or damage or loss of Schwann. . .
SUMM
       . . . in, for example, "Remington's Pharmaceutical Sciences."
       Formulations for parenteral administration may, for example, contain as
       excipients sterile water or saline, polyalkylene
     glycols such as polyethylene glycol, oils of vegetable origin,
       or hydrogenated naphthalenes, biocompatible, biodegradable lactide
       polymer, or polyoxyethylene-polyoxypropylene copolymers may be. . .
L17 ANSWER 65 OF 117 USPATFULL
       Anti-tumor compounds, pharmaceutical compositions, methods for
       preparation thereof and for treatment
ACCESSION NUMBER:
                        97:10043 USPATFULL
TITLE:
                        Anti-tumor compounds, pharmaceutical compositions,
                        methods for preparation thereof and for treatment
                        Ojima, Iwao, Stony Brook, NY, United States
Bombardelli, Ezio, Milan, Italy
INVENTOR(S):
PATENT ASSIGNEE(S):
                        The Research Foundation of State University of New
                        York, Albany, NY, United States (U.S. corporation)
                        Indena SpA Gruppo Inverni Della Beffa, Milan, Italy
                        (non-U.S. corporation)
                             NUMBER
                                         DATE
PATENT INFORMATION:
                        US 5599820 19970204
US 1995-461730 19950605 (8)
                                                                     <--
APPLICATION INFO.:
RELATED APPLN. INFO.:
                        Division of Ser. No. US 1993-40189, filed on 26 Mar
                        1993, now patented, Pat. No. US 5475011
DOCUMENT TYPE:
                        Utility
PRIMARY EXAMINER:
                       Owens, Amelia
LEGAL REPRESENTATIVE: Hoffmann & Baron
NUMBER OF CLAIMS:
EXEMPLARY CLAIM:
                        1
LINE COUNT:
                       1861
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
      US 5599820 19970204
SUMM
      A recent report on clinical trials of Taxol and Taxotere has disclosed
      that Taxol has side effects such as nerve damage,
      muscle pain or disturbances in heart rhythm. Taxotere also has side
      effects. For example, Taxotere provokes mouth sores and a. . .
SUMM
       · · · of pharmaceutically acceptable carriers are, for example,
      manitol, urea, dextrans, lactose, potato and maize starches, magnesium
      stearate, talc, vegetable oils, polyalkylene glycols
       , ethyl cellulose, poly(vinylpyrrolidone), calcium carbonate, ethyl
      oleate, isopropyl myristate, benzyl benzoate, sodium carbonate,
gelatin,
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potassium carbonate, silicic acid, and other. . .

L17 ANSWER 66 OF 117 USPATFULL Glycine receptor antagonist pharmacophore ACCESSION NUMBER: 97:8022 USPATFULL TITLE: Glycine receptor antagonist pharmacophore INVENTOR(S): Cai, Sui X., Irvine, CA, United States Keana, John F. W., Eugene, OR, United States Weber, Eckard, Laguna Beach, CA, United States PATENT ASSIGNEE(S): State of Oregon, Acting by and through the Oregon State Board of Higher Education, Acting for and on Behalf of the Oregon Health Sciences University and the University of Oregon, Eugene, OR, United States (U.S. corporation) Acea Pharmaceuticals, Inc., Irvine, CA, United States (U.S. corporation) The Regents of the University of California, Oakland, CA, United States (U.S. corporation) NUMBER DATE PATENT INFORMATION: US 5597922 19970128 <--APPLICATION INFO.: US 1994-281995 19940729 (8) DOCUMENT TYPE: Utility PRIMARY EXAMINER: Bernhardt, Emily LEGAL REPRESENTATIVE: Sterne, Kessler, Goldstein & Fox, P.L.L.C. NUMBER OF CLAIMS: EXEMPLARY CLAIM: LINE COUNT: 3446 CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5597922 19970128 \cdot . . from a stroke, the compounds of the present invention may be SUMM administered to ameliorate the immediate ischemia and prevent further neuronal damage that may occur from recurrent strokes. . . . oil, or synthetic fatty acid esters, for example, ethyl oleate SUMM or triglycerides or polyethylene glycol-400 (the compounds are soluble in PEG-400). Aqueous injection suspensions may contain substances which increase the viscosity of the suspension include, for example, sodium carboxymethyl cellulose, sorbitol,. . . L17 ANSWER 67 OF 117 USPATFULL Ligands for flt3 receptors ACCESSION NUMBER: 96:82587 USPATFULL TITLE: Ligands for flt3 receptors INVENTOR(S): Lyman, Stewart D., Seattle, WA, United States Beckmann, M. Patricia, Poulsbo, WA, United States PATENT ASSIGNEE(S): Immunex Corporation, United States (U.S. corporation) NUMBER DATE -----PATENT INFORMATION: US 5554512 19960910 <--US 1994-243545 19940511 (8) APPLICATION INFO.: RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1994-209502, filed on 7 Mar 1994, now abandoned which is a continuation-in-part of Ser. No. US 1993-162407, filed on 3 Dec 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-111758, filed on 25 Aug 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-106463, filed

on 12 Aug 1993, now abandoned which is a

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continuation-in-part of Ser. No. US 1993-68394, filed
                         on 24 May 1993, now abandoned
 DOCUMENT TYPE:
                         Utility
 PRIMARY EXAMINER:
                         Walsh, Stephen G.
 ASSISTANT EXAMINER:
                         Spector, Lorraine M.
 LEGAL REPRESENTATIVE:
                         Malaska, Stephen L.
 NUMBER OF CLAIMS:
                         21
 EXEMPLARY CLAIM:
                         1
 LINE COUNT:
                         2004
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.
        US 5554512 19960910
        . . . stimulation is beneficial when specific tissue damage has
 DETD
        occurred to these tissues. As such, flt3-L may be useful in treating
      neurological damage and may be a growth factor for
        nerve cells. It is probable that flt3-L would be useful in in vitro. .
           . . Remington's Pharmaceutical Sciences, 16th ed. 1980, Mack
 DETD
        Publishing Co. In addition, such compositions can contain flt3-L
        complexed with polyethylene glycol (PEG), metal ions, or
        incorporated into polymeric compounds such as polyacetic acid,
       polyglycolic acid, hydrogels, etc., or incorporated into liposomes,
       microemulsions,.
 L17 ANSWER 68 OF 117 USPATFULL
       Heterocyclic derivatives in the treatment of ischaemia and related
       diseases
ACCESSION NUMBER:
                        96:72893 USPATFULL
TITLE:
                        Heterocyclic derivatives in the treatment of ischaemia
                        and related diseases
INVENTOR(S):
                        Pascal, Jean-Claude, Cachan, France
                        McCort, Gary, Paris, France
                        Blondet, Dominique, Paris, France
                        Gellibert, Fran.cedilla.oise, Cachan, France
PATENT ASSIGNEE(S):
                        Syntex Pharmaceuticals, Limited, Maidenhead, England
                        (non-U.S. corporation)
                            NUMBER
                                          DATE
                        -----
PATENT INFORMATION:
                        US 5545645
                                        19960813
                                                                    <--
APPLICATION INFO.:
                        US 1995-401486 19950309 (8)
RELATED APPLN. INFO.:
                        Division of Ser. No. US 1993-45568, filed on 9 Apr
                        1993, now patented, Pat. No. US 5428037
DOCUMENT TYPE:
                        Utility
PRIMARY EXAMINER:
                        Shah, Mukund J.
ASSISTANT EXAMINER:
                        Wong, King Lit
LEGAL REPRESENTATIVE:
                        Heller Ehrman White & McAuliffe
NUMBER OF CLAIMS:
                        10
EXEMPLARY CLAIM:
                        1
LINE COUNT:
                        2655
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
      US 5545645 19960813
SUMM
            . treated by direct neuronal protection, such as ischaemia
      including focal and global ischaemia, cerebral ischaemia including
       ischaemia-induced neurodegeneration, perinatal asphyxia, spinal
    injuries, peripheral nerve ischaemia, peripheral nerve
    damage, head trauma, primary intracerebral hemorrhage,
      encephalopathy, epilepsy or epileptic psychotic symptoms, and
      neurological diseases such as Alzheimer's, Huntington's chorea,
      Parkinsons.
       . . . treated by direct neuronal protection, such as ischaemia
SUMM
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including focal and global ischaemia, cerebral ischaemia including

ischaemia-induced neurodegeneration, perinatal asphyxia, spinal injuries, peripheral nerve ischaemia, peripheral nerve damage, head trauma, primary intracerebral hemorrhage, encephalopathy, epilepsy or epileptic psychotic symptoms, and neurological diseases such as Alzheimer's, Huntington's chorea, Parkinsons. For systemic administration via suppository, traditional binders and SUMM carriers include, for example, polyalkaline glycol or triglycerides [e.g., PEG 1000 (96%) and PEG 4000 (4%)]. Such suppositories may be formed from mixtures containing active ingredients in the range of from about 0.5 wt/%. . L17 ANSWER 69 OF 117 USPATFULL DNA encoding glial mitogenic factors 96:55863 USPATFULL ACCESSION NUMBER: DNA encoding glial mitogenic factors TITLE: Goodearl, Andrew, Chorleywood, United Kingdom INVENTOR(S): Stroobant, Paul, London, England Minghetti, Luisa, Bagnacavallo, Italy Waterfield, Michael, Newbury, United Kingdom Marchioni, Mark, Arlington, MA, United States Chen, Mario S., Arlington, MA, United States Hiles, Ian, London, England PATENT ASSIGNEE(S): Ludwig Institute For Cancer Research, New York, NY, United States (U.S. corporation) Cambridge Neuroscience, Cambridge, MA, United States (U.S. corporation) DATE NUMBER US 5530109 19960625 US 1993-36555 19930324 (8) PATENT INFORMATION: APPLICATION INFO.: RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1992-965173, filed on 23 Oct 1992, now abandoned Ser. No. Ser. No. US 1992-940389, filed on 3 Sep 1992, now abandoned Ser. No. Ser. No. US 1992-907138, filed on 30 Jun 1992, now abandoned And Ser. No. US 1992-863703, filed on 3 Apr 1992, now abandoned DATE NUMBER ______ GB 1991-7566 19910410 PRIORITY INFORMATION: DOCUMENT TYPE: Utility Walsh, Stephen G. PRIMARY EXAMINER: ASSISTANT EXAMINER: Cermak, Shelly Guest LEGAL REPRESENTATIVE: Felfe & Lynch; Butler, Gregory B. NUMBER OF CLAIMS: 12 EXEMPLARY CLAIM: 89 Drawing Figure(s); 78 Drawing Page(s) NUMBER OF DRAWINGS: LINE COUNT: 3401 CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5530109 19960625 SUMM Included in the invention as well, are methods for treatment when the

nervous system; or damage or loss of Schwann. . . .

SUMM . . . in, for example, "Remington's Pharmaceutical Sciences."

Formulations for parenteral administration may, for example, contain as excipients sterile water or saline, polyalkylene glycols such as polyethylene glycol, oils of vegetable origin,

neurodegenerative disorders; demyelination in peripheral or central

condition involves peripheral nerve damage; nerve damage in the central nervous system;

or hydrogenated naphthalenes, biocompatible, biodegradable lactide polymer, or polyoxyethylene-polyoxypropylene copolymers may be. .

L17 ANSWER 70 OF 117 USPATFULL

TI Method for production and purification or recombinant Apolipoprotein E from bacteria

ACCESSION NUMBER:

96:50772 USPATFULL

TITLE:

INVENTOR(S):

Method for production and purification or recombinant

Apolipoprotein E from bacteria Lifshitz, Ruth, Rehovot, Israel

Fischer, Meir, Rehovot, Israel Greenman, Benjamin, Rehovot, Israel Bartfeld, Daniel, Ontario, Canada

PATENT ASSIGNEE(S):

Bio-Technology General Corp., Iselin, NJ, United

States

(U.S. corporation)

NUMBER DATE

PATENT INFORMATION:

US 5525472 19960611 <--

APPLICATION INFO.:

US 1994-333872 19941103 (8)

RELATED APPLN. INFO.:

Continuation of Ser. No. US 1993-59889, filed on 10

May

1993, now abandoned which is a continuation of Ser.

No.

US 1991-721159, filed on 26 Jun 1991, now abandoned

Utility

PRIMARY EXAMINER: LEGAL REPRESENTATIVE: Walsh, Stephen G. White, John P.

NUMBER OF CLAIMS:

6 1

EXEMPLARY CLAIM: NUMBER OF DRAWINGS:

8 Drawing Figure(s); 4 Drawing Page(s)

LINE COUNT:

DOCUMENT TYPE:

1451

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 5525472 19960611

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DETD . . . the ApoE analog is suspended in a buffer containing non-ionic detergent. Preferably the non-ionic detergent is Emulphogen.sup.R -BC720

(Sigma) or **PEG** (9-10) p-t- octylphenol which is sold under the tradename Triton.sup.R X-100 (Merck), designated Triton.sup.R. Triton.sup.R was used at a concentration. . .

DETD Additionally, the invention provides a method of treating a subject suffering from **neuronal injury** which comprises administering to the subject an amount of ApoE or analog thereof effective to promote nerve development and regeneration.

DETD 2. Treatment of Neuronal Injury

L17 ANSWER 71 OF 117 USPATFULL

TI Substituted imidazolyl-alkyl-piperazine and -diazepine derivatives

ACCESSION NUMBER:

96:39021 USPATFULL

TITLE:

Substituted imidazolyl-alkyl-piperazine and -diazepine

derivatives

INVENTOR(S):

Pascal, Jean C., Cachan, France

Lee, Chi-Ho, Palo Alto, CA, United States Alps, Brian J., Linlithgow, Scotland

Pinhas, Henri, Paris, France

Whiting, Roger L., Los Altos, CA, United States MacFarlane, Calum B., Linlithgow, Scotland Beranger, Serge, Bretigny-Sur-Cedres, France

PATENT ASSIGNEE(S):

Syntex Pharmaceuticals, Ltd., Maidenhead, England

(non-U.S. corporation)

NUMBER DATE _____

US 5514800 PATENT INFORMATION: 19960507

US 1993-124518 APPLICATION INFO.: 19930920 (8)

Division of Ser. No. US 1991-688193, filed on 19 Apr RELATED APPLN. INFO.:

1991, now patented, Pat. No. US 5276034 which is a division of Ser. No. US 1988-260969, filed on 21 Oct 1988, now patented, Pat. No. US 5043447 which is a continuation-in-part of Ser. No. US 1987-42181, filed

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on 24 Apr 1987, now patented, Pat. No. US 4829065

DOCUMENT TYPE: Utility

Tsang, Cecilia PRIMARY EXAMINER:

Heller Ehrman White & McAuliffe LEGAL REPRESENTATIVE:

NUMBER OF CLAIMS: 10 EXEMPLARY CLAIM: 2247 LINE COUNT:

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5514800 19960507 PΤ SUMM

diseases treated by direct neuronal protection, such as ischemia including focal and global ischemia, spinal injuries

, head trauma, and neurological diseases such as Alzheimer's and Huntington's chorea;

SUMM diseases treated by direct neuronal protection, such as ischemia including focal and global ischemia, spinal injuries , head trauma, and neurological diseases such as Alzheimer's and Huntington's chorea;

SUMM diseases treated by direct neuronal protection, such as ischemia including focal and global ischemia, spinal injuries , head trauma, and neurological diseases such as Alzheimer's and Huntington's chorea;

SUMM For systemic administration via suppository, traditional binders and carriers include, for example, polyalkaline glycol or triglycerides [e.g., PEG 1000 (96%) and PEG 4000 (4%)]. Such suppositories may be formed from mixtures containing active ingredients in the range of from about 0.5 wt/%. . .

L17 ANSWER 72 OF 117 USPATFULL

Glycine receptor antagonists and the use thereof

ACCESSION NUMBER: 96:38902 USPATFULL

TITLE: Glycine receptor antagonists and the use thereof INVENTOR(S): Weber, Eckard, Laguna Beach, CA, United States Keana, John F. W., Eugene, OR, United States

PATENT ASSIGNEE(S): The State of Oregon, acting by and through The Oregon State Board of Higher Education, acting for and on behalf of The Oregon Health Sciences University, Eugene, OR, United States (U.S. corporation)

The University of Oregon, Eugene, OR, United States

(U.S. corporation)

The Regents of the University of California, Oakland,

CA, United States (U.S. corporation)

NUMBER ______

PATENT INFORMATION: APPLICATION INFO .:

US 5514680 19960507 US 1993-148259 19931105 (8)

RELATED APPLN. INFO .: Continuation-in-part of Ser. No. US 1993-69274, filed

on 28 May 1993, now abandoned which is a

continuation-in-part of Ser. No. US 1992-995167, filed

on 22 Dec 1992, now abandoned which is a

continuation-in-part of Ser. No. US 1992-903080, filed

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on 22 Jun 1992, now abandoned
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DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Hollrah, Glennon H. ASSISTANT EXAMINER: Cebulak, Mary C.

LEGAL REPRESENTATIVE: Sterne, Kessler, Goldstein & Fox

NUMBER OF CLAIMS: 26 EXEMPLARY CLAIM:

45 Drawing Figure(s); 45 Drawing Page(s) NUMBER OF DRAWINGS:

LINE COUNT: 7435

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5514680 19960507

. . . from a stroke, the compounds of the present invention may be DETD administered to ameliorate the immediate ischemia and prevent further

neuronal damage that may occur from recurrent strokes.

. . . oil, or synthetic fatty acid esters, for example, ethyl oleate or triglycerides or polyethylene glycol-400 (the compounds are soluble in PEG-400). Aqueous injection suspensions may contain substances which increase the viscosity of the suspension include, for example, sodium carboxymethyl cellulose, sorbitol,. . .

Also conducted were dose-response experiments i.v. using a DETD TRIS/TWEEN-80/PEG-400 formulation as a vehicle for the drug. The ED.sub.50 value (5-6 mg/kg) for 5-NO.sub.2 -6,7-Cl.sub.2 --QX in this formulation was.

. . extended to both locomotor activity and radial arm maze DETD performance. Consistent with this robust behavioral protection, 5-chloro-7-trifluoromethyl-1,4-dihydroquinoxaline-2,3-dione also protected against neuronal damage.

A 5 mg/ml solution of 5-nitro-6,7-dichloro-1,4-dihydroquinoxaline-2,3-DETD dione was prepared by dissolving the 5-nitro-6,7-quinoxaline-2,3-dione in an aqueous solution containing 10% polyethyleneglycol 400 (

PEG-400), 0.45% TWEEN-80 and 0.18M TRIS (Tromethamine) to give a final concentration of 5 mg/ml of 5-nitro-6,7-dichloro-1,4dihydroquinoxaline-2,3-dione. The 5-nitro-6,7-dichloro-1,4dihydroquinoxaline-2,3-dione readily dissolved. . . least 1-2 years. A 10 mg/ml solution of 5-nitro-6,7-dichloro-1,4-dihydroquinoxaline-2,3dione was prepared by dissolving the compound in a solution containing (50% PEG-400,) 0.5% TWEEN-80 and 0.1M TRIS (Tromethamine). The 5-nitro-6,7-dichloro-1,4-dihydroquinoxaline-2,3-dione readily dissolved in this solution by warming to 60.degree.-100.degree. C. The solution. . this solution will be stable for at least 1-2 years. A 5 mg/ml solution of 5-nitro-6,7-dichloro-1,4-dihydroquinoxaline-2,3-dione was

also prepared without PEG-400 by dissolving the compound in a solution containing 0.05M TRIS (Tromethamine), 0.5% TWEEN-80 and 5% glucose. The solution was sterilized.

A 10 mg/ml solution of

5-chloro-7-trifluoromethyl-1, 4-dihydroquinoxaline-

2,3-dione was prepared by dissolving the compound in 0.1M bis-tris-propane, 50% PEG-400 or propyleneglycol, 0.75% TWEEN-80. The compound dissolved readily by warming in a boiling water bath. The solution was autoclaved and. .

ANSWER 73 OF 117 USPATFULL

Process for producing flowable osteogenic composition containing demineralized bone particles

96:34171 USPATFULL ACCESSION NUMBER:

Process for producing flowable osteogenic composition TITLE:

containing demineralized bone particles

Prewett, Annamarie B., Little Silver, NJ, United INVENTOR(S):

States

Stikeleather, Roger C., Doylestown, PA, United States Osteotech, Inc., Shrewsbury, NJ, United States (U.S. PATENT ASSIGNEE(S):

corporation)

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DATE
                            NUMBER
                        ______
                       US 5510396 19960423 <--
US 1994-208432 19940309 (8)
Division of Ser. No. US 1993-119882, filed on 10 Sep
PATENT INFORMATION:
APPLICATION INFO.:
RELATED APPLN. INFO.:
                       1993, now patented, Pat. No. US 5314476 which is a
                       continuation of Ser. No. US 1992-830934, filed on 4
Feb
                       1992, now abandoned
DOCUMENT TYPE:
                       Utility
PRIMARY EXAMINER:
                       Cain, Edward J.
LEGAL REPRESENTATIVE:
                       Dilworth & Barrese
NUMBER OF CLAIMS:
                       20
EXEMPLARY CLAIM:
LINE COUNT:
                        504
      US 5510396 19960423
PΙ
       . . derivatives of the foregoing. Specific polyhydroxy compounds
SUMM
      include ethylene glycol, diethylene glycol, triethylene glycol,
       1,2-propanediol, glycerol, trimethylolethane, trimethylolpropane,
       erythritol, pentaerythritol, polyalkylene glycols
       such as the polyethylene glycols, xylitol, sorbitol, mannitol,
dulcitol,
      arabinose, xylose, ribose, adonitol, arabitol, rhamose, inositol,
       fructose, galactose, glucose, mannose,. .
       . . fixation, tumor surgery, e.g., deficit filling, discectomy,
SUMM
      laminectomy, excision of spinal cord tumors, anterior cervical and
      thoracic operations, repair of spinal injuries,
       scoliosis, lordosis and kyphosis treatments, intermaxillary fixation of
       fractures, mentoplasty, temporomandibular joint replacement, alveolar
       ridge augmentation and reconstruction, inlay bone. . .
L17 ANSWER 74 OF 117 USPATFULL
      Cysteine protease and serine protease inhibitors
                       96:21096 USPATFULL
ACCESSION NUMBER:
TITLE:
                       Cysteine protease and serine protease inhibitors
INVENTOR(S):
                       Mallamo, John P., Glenmore, PA, United States
                       Bihovsky, Ron, Wynnewood, PA, United States
                       Chatterjee, Sankar, Wynnewood, PA, United States
                       Tripathy, Rabindranath, Pennsville, NJ, United States
PATENT ASSIGNEE(S):
                       Cephalon, Inc., West Chester, PA, United States (U.S.
                       corporation)
                           NUMBER DATE
                       US 5498616 19960312
PATENT INFORMATION:
                                                                    <--
                       US 1994-334249 19941104 (8)
APPLICATION INFO.:
DOCUMENT TYPE:
                       Utility
                       Raymond, Richard L.
PRIMARY EXAMINER:
                       Woodcock Washburn Kurtz Mackiewicz & Norris
LEGAL REPRESENTATIVE:
NUMBER OF CLAIMS:
                       18
EXEMPLARY CLAIM:
                       1
LINE COUNT:
                       1271
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
      US 5498616 19960312
PΤ
       . . . calpain family of cysteine proteases has been implicated in
SUMM
      many diseases and disorders, including neurodegeneration, stroke,
      Alzheimer's disease, amyotrophy, motor neuron damage
       , acute central nervous system injury, muscular dystrophy, bone
       resorption, platelet aggregation, cataracts and inflammation. Calpain I
```

has been implicated in.

. . . Sciences (Mack Pub. Co., Easton, Pa., 1980). Formulations for SUMM parenteral administration may contain as common excipients sterile

water

or saline, polyalkylene glycols such as polyethylene glycol, oils of vegetable origin, hydrogenated naphthalenes and the like. In particular, biocompatible, biodegradable lactide polymer, lactide/glycolide. .

L17 ANSWER 75 OF 117 USPATFULL

Microorganism antigen extraction methods

ACCESSION NUMBER:

96:16881 USPATFULL

TITLE:

Microorganism antigen extraction methods

INVENTOR(S):

Bogart, Gregory R., Berthoud, CO, United States Bilodeau, Robert J., Arvada, CO, United States Ostroff, Rachel M., Westminster, CO, United States Steaffens, Jeffrey W., Louisville, CO, United States

PATENT ASSIGNEE(S):

Biostar, Inc., Boulder, CO, United States (U.S.

corporation)

NUMBER DATE ______

PATENT INFORMATION:

US 5494801 19960227

APPLICATION INFO.:

US 1993-162401 19931203 (8)

DOCUMENT TYPE:

Utility

PRIMARY EXAMINER:

Woodward, Michael P. Stucker, Jeffrey

ASSISTANT EXAMINER: LEGAL REPRESENTATIVE: Lyon & Lyon

NUMBER OF CLAIMS:

44

EXEMPLARY CLAIM: NUMBER OF DRAWINGS:

1 1 Drawing Figure(s); 1 Drawing Page(s)

LINE COUNT:

1153

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5494801 19960227

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SUMM

. . . swab. Reagents are held within the tube by inert binders or carriers such as dextran, polyacrylamide, polyacrylic acid, polyvinyl alcohol, PEG, PEO, PVP, guar gum, caboxymethylcellulose,

hydroxyethyl cellulose, methyl cellulose, algin, carrageenan, and xanthan gum.

SUMM

26% to 50%; and 30% of the infected infants will develop meningitis. Of the latter group, 50% will suffer permanent

neurological damage. Infection with GBS is estimated

to cost the U.S. alone over \$500 million per year in health care.

L17 ANSWER 76 OF 117 USPATFULL

Azepine synthesis via a diels-alder reaction

ACCESSION NUMBER:

95:112616 USPATFULL

TITLE: INVENTOR(S): Azepine synthesis via a diels-alder reaction Keana, John F. W., Eugene, OR, United States

Guzikowski, Anthony P., Eugene, OR, United States Nogales, Daniel F., Nampa, ID, United States

Cai, Sui X., Irvine, CA, United States

PATENT ASSIGNEE(S):

State of Oregon, acting by and through The Oregon

State

Board of Higher Education, acting for and on behalf of

The Oregon health Sciences University and The

University of Oregon, Eugene, OR, United States (U.S.

corporation)

Acea Pharmaceuticals, Inc., Menlo Park, CA, United

States (U.S. corporation)

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NUMBER
                                        DATE
                       US 5476933 19951219
                                                                  <---
PATENT INFORMATION:
APPLICATION INFO.:
                       US 1994-341154 19941116 (8)
DOCUMENT TYPE:
                       Utility
                       Bond, Robert T.
PRIMARY EXAMINER:
                       Sterne, Kessler, Goldstein & Fox
LEGAL REPRESENTATIVE:
NUMBER OF CLAIMS:
EXEMPLARY CLAIM:
LINE COUNT:
                       5077
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
      US 5476933 19951219
         . . from a stroke, the compounds of the present invention may be
      administered to ameliorate the immediate ischemia and prevent further
    neuronal damage that may occur from recurrent strokes.
      . . . oil, or synthetic fatty acid esters, for example, ethyl oleate
      or triglycerides or polyethylene glycol-400 (the compounds are soluble
       in PEG-400). Aqueous injection suspensions may contain
      substances which increase the viscosity of the suspension include, for
       example, sodium carboxymethyl cellulose, sorbitol,. . .
L17 ANSWER 77 OF 117 USPATFULL
      Anti-tumor compounds, pharmaceutical compositions, methods for
      preparation thereof and for treatment
ACCESSION NUMBER: 95:110459 USPATFULL
                       Anti-tumor compounds, pharmaceutical compositions,
TITLE:
                     methods for preparation thereof and for treatment
                       Ojima, Iwao, Stony Brook, NY, United States
INVENTOR(S):
                       Bombardelli, Ezio, Milan, Italy
                       The Research Foundation of State University of New
PATENT ASSIGNEE(S):
                       York, Albany, NY, United States (U.S. corporation)
                                       DATE
                           NUMBER
PATENT INFORMATION:
                       US 5475011 19951212
                                                                  <---
                      US 1993-40189 19930326 (8)
APPLICATION INFO.:
DOCUMENT TYPE:
                      Utility
PRIMARY EXAMINER: Ivy, Warren ASSISTANT EXAMINER: Owens, A. A.
                      Ivy, Warren C.
LEGAL REPRESENTATIVE: Hoffmann & Baron
NUMBER OF CLAIMS:
EXEMPLARY CLAIM:
LINE COUNT:
                      1875
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
      US 5475011 19951212
PΙ
      A recent report on clinical trials of Taxol and Taxotere has disclosed
SUMM
      that Taxol has side effects such as nerve damage,
      muscle pain or disturbances in heart rhythm. Taxotere also has side
       effects. For example, Taxotere provokes mouth sores and a. . .
       . . . of pharmaceutically acceptable carriers are, for example,
SUMM
      manitol, urea, dextrans, lactose, potato and maize starches, magnesium
       stearate, talc, vegetable oils, polyalkylene glycols
       , ethyl cellulose, poly(vinylpyrrolidone), calcium carbonate, ethyl
       oleate, isopropyl myristate, benzyl benzoate, sodium carbonate,
gelatin,
       potassium carbonate, silicic acid, and other. . .
L17 ANSWER 78 OF 117 USPATFULL
       1,2,3,4-tetrahydroquinoline-2,3,4-trione-3 or 4-oximes and the use
       thereof
```

95:110455 USPATFULL

ACCESSION NUMBER:

1,2,3,4-tetrahydroguinoline-2,3,4-trione-3 or 4-oximes TITLE:

and the use thereof

Caı, Sui X., Irvine, CA, United States INVENTOR(S):

Keana, John F. W., Eugene, OR, United States Weber, Eckard, Laguna Beach, CA, United States The Regents of the University of California, Oakland,

PATENT ASSIGNEE(S):

CA, United States (U.S. corporation)

State of Oregon, acting by and through the Oregon

State

Board of Higher Education, acting for and on behalf of

the Oregon Health Sciences University and the

University of Oregon, Eugene, OR, United States (U.S.

corporation)

NUMBER DATE

PATENT INFORMATION:

US 5475007 19951212

APPLICATION INFO.:

US 1993-69005 19930528 (8)

DOCUMENT TYPE: Utility

Daus, Donald G. PRIMARY EXAMINER:

Sterne, Kessler, Goldstein & Fox LEGAL REPRESENTATIVE:

NUMBER OF CLAIMS: 10 EXEMPLARY CLAIM: 1 LINE COUNT: 1934

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5475007 19951212

<--

. . from a stroke, the compounds of the present invention may be $% \left(\frac{1}{2}\right) =\frac{1}{2}\left(\frac{1}{2}\right) =\frac{1}{2}\left($ administered to ameliorate the immediate ischemia and prevent further neuronal damage that may occur from recurrent strokes.

. . . oil, or synthetic fatty acid esters, for example, ethyl oleate or triglycerides or polyethylene glycol-400 (the compounds are soluble in PEG-400). Aqueous injection suspensions may contain

substances which increase the viscosity of the suspension include, for example, sodium carboxymethyl cellulose, sorbitol,. . .

L17 ANSWER 79 OF 117 USPATFULL

Selected protein kinase inhibitors for the treatment of neurological TΤ disorders

ACCESSION NUMBER:

PATENT ASSIGNEE(S):

APPLICATION INFO.:

95:95012 USPATFULL

TITLE:

Selected protein kinase inhibitors for the treatment

of

neurological disorders

Lewis, Michael E., West Chester, PA, United States INVENTOR(S): Kauer, James C., Kennett Square, PA, United States

> Neff, Nicola, Wallingford, PA, United States Roberts-Lewis, Jill, West Chester, PA, United States

Murakata, Chikara, Hachioji, Japan Saito, Hiromitsu, Mishima, Japan Matsuda, Yuzuru, Koganei, Japan

Glicksman, Marcie A., Swarthmore, PA, United States Cephalon, Inc., West Chester, PA, United States (U.S.

corporation)

Kyowa Hakko Kogyo Co., Ltd., Tokyo, Japan (non-U.S.

corporation)

NUMBER DATE

PATENT INFORMATION:

US 5461146 19951024 US 1993-96561 19930722 (8)

Continuation-in-part of Ser. No. US 1992-920102, filed RELATED APPLN. INFO.:

on 24 Jul 1992, now abandoned

```
Utility
DOCUMENT TYPE:
PRIMARY EXAMINER:
                       Datlow, Philip I.
                       Fish & Richardson
LEGAL REPRESENTATIVE:
NUMBER OF CLAIMS:
EXEMPLARY CLAIM:
                       1,2,3,4
NUMBER OF DRAWINGS:
                       20 Drawing Figure(s); 14 Drawing Page(s)
LINE COUNT:
                       1425
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
      US 5461146 19951024
      . . . Sciences (Mack Pub. Co, Easton, Pa., 1980). Formulations for
DETD
      parenteral administration may contain as common excipients sterile
water
      or saline, polyalkylene glycols such as polyethylene
       glycol, oils of vegetable origin, hydrogenated naphthalenes and the
       like. In particular, biocompatable, biodegradable lactide polymer,
       lactide/glycolide.
      The effect of K-252a or its derivatives on kainate-induced
DETD
    neuronal damage was evaluated as follows: Adult male
       or female Sprague-Dawley rats (175-250 g) were anesthetized with
      Nembutal (50 mg/kg, ip) and.
L17 ANSWER 80 OF 117 USPATFULL
      Regimen method of mediating neuronal damage using nitroglycerine
ACCESSION NUMBER:
                       95:88494 USPATFULL
                       Regimen method of mediating neuronal damage using
TITLE:
                       nıtroglycerine
INVENTOR(S):
                       Lipton, Stuart A., Newton, MA, United States
PATENT ASSIGNEE(S):
                       The Children's Medical Center Corporation, Boston, MA,
                       United States (U.S. corporation)
                                        DATE
                            NUMBER
                       US 5455279 19951003
US 1993-25028 19930302 (8)
PATENT INFORMATION:
APPLICATION INFO.:
                       Continuation-in-part of Ser. No. US 1992-949342, filed
RELATED APPLN. INFO.:
                       on 22 Sep 1992, now patented, Pat. No. US 5234956
which
                        is a continuation of Ser. No. US 1991-688965, filed on
                        19 Apr 1991, now abandoned
DOCUMENT TYPE:
                       Utility
PRIMARY EXAMINER:
                       Cintins, Marianne M.
                       Criares, T. J.
ASSISTANT EXAMINER:
LEGAL REPRESENTATIVE: Fish & Richardson
NUMBER OF CLAIMS:
                       1.1
EXEMPLARY CLAIM:
NUMBER OF DRAWINGS:
                       12 Drawing Figure(s); 7 Drawing Page(s)
LINE COUNT:
                        960
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       US 5455279 19951003
PΙ
       I have discovered that certain compounds protect neurons against NMDA
SUMM
       receptor-mediated neuronal damage. Specifically,
       nitroglycerin, nitroprusside, and their nitroso-compound derivatives
       provide such protection. Thus, one aspect of the invention features a
       method for decreasing NMDA receptor complex-mediated neuronal
     damage in a mammal by administering one of the above-described
       compounds to the mammal, in a concentration effective to decrease such.
       . . . However, it appears that oxidation of the thiol group(s) of
SUMM
the
       NMDA receptor's redox modulatory site protect against NMDA
```

receptor-mediated neuronal damage. It is also known

```
that the active species of nitroglycerin and nitroprusside is nitric
       oxide or related NO redox species..sup.1. .
      A second aspect of the invention features a method for decreasing NMDA
SUMM
      receptor complex-mediated neuronal damage by
       administering a nitroso-compound, in a concentration effective to cause
       neuroprotection -- e.g., a decrease in such damage. Without wishing to.
      By "NMDA receptor-mediated neuronal damage" is meant
SUMM
       any neuronal injury which is associated with
      stimulation or co-stimulation of the NMDA receptor-channel complex, a
       receptor-channel complex which is found on a. .
       . . (most probably a related redox species such as an NO.sup.+ or
SUMM
       NO.sup.- equivalent) upon administration to a mammal to decrease
     neuronal damage or injury. For convenience, I have
       also used the less precise term "NO-generating compound" to include
       compounds that produce the.
SUMM
       . . . the desired neuroprotective effect. Accordingly, the fourth
       aspect features administering a nitroso compound capable of protecting
       against NMDA receptor complex-mediated neuronal injury
       , continuously over an extended period with gradually escalating
dosage,
      beginning at a dosage level which does not substantially reduce the.
       . . or it can be used independently, particularly to treat
SHMM
      neurological manifestations of infection with HIV or of ALS.
       Polyethylene glycol (PEG) is used to enhance absorption into
       the central nervous system (CNS) and efficacy of SOD and/or catalase.
An
       SOD mimic, . . . polysaccharide of Coriolus versicolor QUEL, termed
       "PS-K", may also be effective by parenteral or oral routes of
       administration, especially with PEG to enhance CNS absorption,
       and such mimics may be substituted for SOD in this aspect of the
       invention. See Kariya.
DETD
       The present invention is based on the finding that the compounds
       nitroprusside and nitroglycerin decrease NMDA receptor complex-mediated
     neuronal damage (see below). This neuroprotection may
      be due to nitrosation or oxidation of the NMDA receptor at the redox
      modulatory site,.
DETD
       . . . one or more glutamate-related compounds is associated with
many
       neurodegenerative disorders (e.g., those listed above). In addition to
       qlutamate itself, neuronal injury may result from
       stimulation of the NMDA receptor-channel complex by other excitatory
       amino acids, such as aspartate, quinolinate, homocysteic acid, . . .
DETD
       . . . second aspect of the invention (i.e., nitroso-compounds or
       NO-generating compounds and their derivatives) may be tested for
       efficacy in decreasing neuronal damage using the
       assays described below--i.e. in assays of NMDA-evoked ionic current
       (see, e.g., PCT WO 91/02810), in assays of NMDA-. .
       The following examples illustrate compounds useful in the method of the
       invention and their efficacy in reducing neuronal
     damage. These examples are provided to illustrate the invention
       and should not be construed as limiting.
       We found that either NTG or SNP ameliorated neuronal
     injury engendered by the addition of NMDA after exposure to DTT
       (FIGS. 6A and 6B). The latter compound was added to. . .
DETD
       . . . liberation of NO. (t.sub.1/2(pH 7.4) of S-nitrosocysteine
       .about.30 s) for reaction with endogenous O.sub.2..sup.- to form
       peroxynitrite (ONOO-) with subsequent neuronal damage
```

To prevent neuronal damage, compounds of the

DETD

```
invention may be administered by any of a number of routes in an amount
      sufficient to attenuate. . .
       . . be an effective neuroprotective agent by the assays described
DETD
      herein, are administered as above, at a dosage suitable to reduce
    neuronal damage, or NMDA evoked ionic current or
      increased [Ca.sup.2+ ]i. Generally, such compounds are administered in
      dosages ranging from 0.01 mg-60.
DETD
       . . . is predictive of useful NO-conjugate dosage. Dosages may be
      divided. Treatment may be repeated as necessary to prevent or alleviate
    neurological injury. It is desirable to maintain
      levels of NO or related redox species in the brain of 1 nM to 500.
DETD
              and other neurological manifestations of the AIDS virus (HIV-1
      or HIV-2). The method may also be used for reduction of neuronal
    damage resulting from infection with other viruses, such as
      measles, which cause damage to the nervous system. Other diseases
listed
      above.
DETD
      The method described herein is useful for reducing neuronal
    injury in any mammal having NMDA receptors. Treatment of
    neuronal damage in humans is the preferred utility;
      but the method may also be employed successfully for veterinary
      purposes. The NO-generating compound. . . polysaccharide of Coriolus
      versicolor QUEL, termed "PS-K", may also be effective by parenteral or
      oral routes of administration, especially with PEG to enhance
      CNS absorption. PQQ (pyrroloquinoline quinone-see U.S. Pat. No.
      5,091,391, hereby incorporated by reference or PQQ's derivative esters
       . . . nitroso-compounds) would limit NO. production (e.g., nitric
DETD
      oxide synthase (NOS) inhibitors). Such treatment would avoid
      peroxynitrite (ONOO.sup.-) formation and hence neuronal
    injury, e.g., contribution to the AIDS dementia complex and
      other neurological manifestations of AIDS. These agents are listed in
      Table 3.
DETD
      Acute Neurologic Disorders with Neuronal Damage
      Thought to be Mediated at Least in Part by Excitatory Amino Acids*
      Chronic Neurodegenerative Diseases with Neuronal
DETD
    Damage Thought or Proposed to be Mediated at Least in Part by
      Excitatory Amino Acids.*
L17 ANSWER 81 OF 117 USPATFULL
      Heterocyclic derivatives in the treatment of Ischaemia and related
      diseases
ACCESSION NUMBER:
                       95:58140 USPATFULL
                       Heterocyclic derivatives in the treatment of Ischaemia
TITLE:
                       and related diseases
INVENTOR(S):
                       Pascal, Jean-Claude, Cachan, France
                       McCort, Gary, Paris, France
                       Blondet, Dominique, Paris, France
                       Gellibert, Francoise, Cachan, France
PATENT ASSIGNEE(S):
                       Syntex Pharmaceuticals, Ltd., Maidenhead, England
                        (non-U.S. corporation)
                            NUMBER
                       ______
                       US 5428037 19950627
US 1993-45568 19930409 (8)
                                                                   <--
PATENT INFORMATION:
APPLICATION INFO.:
DOCUMENT TYPE:
                       Utility
PRIMARY EXAMINER:
                      Tsang, Cecilia
LEGAL REPRESENTATIVE: Lewis, Brian
```

NUMBER OF CLAIMS:

38

EXEMPLARY CLAIM: 1
LINE COUNT: 2754

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 5428037 19950627

SUMM . . . treated by direct neuronal protection, such as ischaemia including focal and global ischaemia, cerebral ischaemia including ischaemia-induced neurodegeneration, perinatal asphyxia, spinal

injuries, peripheral nerve ischaemia, peripheral nerve damage, head trauma, primary intracerebral hemorrhage,

encephalopathy, epilepsy or epileptic psychotic symptoms, and neurological diseases such as Alzheimer's, Huntington's chorea, Parkinsons. . .

SUMM . . . treated by direct neuronal protection, such as ischaemia including focal and global ischaemia, cerebral ischaemia including ischaemia-induced neurodegeneration, perinatal asphyxia, spinal

injuries, peripheral nerve ischaemia, peripheral nerve
damage, head trauma, primary intracerebral hemorrhage,

encephalopathy, epilepsy or epileptic psychotic symptoms, and neurological diseases such as Alzheimer's, Huntington's chorea, Parkinsons. . .

SUMM For systemic administration via suppository, traditional binders and carriers include, for example, polyalkaline glycol or triglycerides [e.g., PEG 1000 (96%) and PEG 4000 (4%)]. Such suppositories may be formed from mixtures containing active ingredients in the range of from about 0.5 wt.. . .

L17 ANSWER 82 OF 117 USPATFULL

TI Screening method for neuroprotective compounds

ACCESSION NUMBER:

95:52264 USPATFULL

TITLE:

Screening method for neuroprotective compounds

INVENTOR(S):

Miljanich, George P., Redwood City, CA, United States Bitner, Robert S., Mountain View, CA, United States Bowersox, Stephen S., Menlo Park, CA, United States

Fox, James A., Palo Alto, CA, United States

Valentino, Karen L., San Carlos, CA, United States Yamashiro, Donald H., San Francisco, CA, United States

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PATENT ASSIGNEE(S):

Neurex Corporation, Menlo Park, CA, United States

(U.S.

corporation)

NUMBER DATE

PATENT INFORMATION: US 5424218 19950613 APPLICATION INFO.: US 1993-147714 19931104 (8)

APPLICATION INFO.: US 1993-147/14 19931104 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1992-855269, filed on 23 Mar 1992, now patented, Pat. No. US 5264371 which is a division of Ser. No. US 1990-561766, filed on 2 Aug 1990, now patented, Pat. No. US 5189020 which is a continuation-in-part of Ser. No. US 1989-440094, filed

on 22 Nov 1989, now patented, Pat. No. US 5051403

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Russel, Jeffrey E.

LEGAL REPRESENTATIVE: Dehlinger, Peter J.; Stratford, Carol A.

NUMBER OF CLAIMS: 3 EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 22 Drawing Figure(s); 12 Drawing Page(s)

LINE COUNT: 1941

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 5424218 19950613

DETD Co-owned U.S. patent application for "Method of Treating Ischemia-Related Neuronal Damage," Ser. No. 440,094,

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filed Nov. 22, 1989, now U.S. Pat. No. 5,051,403, describes a method of
       reducing neuronal damage related to ischemia, by
       administering OCT peptides which have certain binding and/or inhibitory
       properties. The properties which were found to.
       In vitro and in vivo studies reported in the above-cited patent
DETD
       application for "Method of Treating Ischemia-Related Neuronal
    Damage, " demonstrate a strong correlation, between (a) high
       binding affinity to synaptosomal membranes, (b) inhibition of
       voltage-gated calcium ion currents and neurotransmitter release
       selectively in neuronal cells, and (c) ability to reduce
     neuronal damage in ischemia-related injury, such as
       stroke. The mechanism of neural protection by high-affinity OCT
peptides
       presumably involves inhibition of voltage-gated. . . and the
       consequent release of neurotransmitters from the cells. This mechanism
       of OCT protection is consistent with the finding that neuronal
     damage in ischemia-related injury is associated with elevated
       intracellular calcium levels (Deshpande et al.).
       . . . effective inhibitors of voltage-gated calcium currents in
DETD
       neuronal cells, and that such compounds, in turn, are useful for
       reducing ischemia-related neuronal damage, such as
       caused by stroke. This model is the basis of the screening method of
the
       invention.
       . . . the invention facilitates the screening of effective
DETD
       neuroprotective compounds. One criterion for an effective
       neuroprotective compound, for use in reducing neuronal
     damage in ischemia-related injury, is the ability to inhibit the
       spread of neuronal damage from the site of injury.
       Evidence indicates that the spread of damage in ischemia-related injury
       is due, at least in. . .
       In another aspect, the present invention provides a treatment method
DETD
for
       reducing neuronal damage related to an ischemic
       condition in a human patient, by administration of a pharmaceutically
       effective amount of a compound selected.
       . . to OCT binding sites in neuronal tissue and (b) selective
DETD
       inhibition of calcium channel currents and neurotransmitter release in
       reducing neuronal damage in ischemia-related in
       jury. Based on the apparent mechanism of action of the OCT peptides, it
       can be predicted that. .
       . . . through 0.6% polyethyleneimine treated GF/C filters
DETD
(Millipore)
       on a Millipore filtration unit. Protein bound [.sup.125 I]MVIIA OCT
       present in the PEG precipitate was determined by gamma
       counting. FIG. 7 illustrates displacement of [.sup.125 I]-MVIIA OCT
       binding by unlabeled MVIIA OCT in. . .
L17 ANSWER 83 OF 117 USPATFULL
       Method for performing a gastric wrap of the esophagus for use in the
       treatment of esophageal reflux
ACCESSION NUMBER:
                        95:29134 USPATFULL
                        Method for performing a gastric wrap of the esophagus
TITLE:
                        for use in the treatment of esophageal reflux
                        Harrison, Michael R., San Francisco, CA, United States
INVENTOR(S):
                        Jennings, Russell W., Pacifica, CA, United States
                        Flake, Alan W., San Francisco, CA, United States
The Regents of the University of California, Oakland,
PATENT ASSIGNEE(S):
```

NUMBER DATE

CA, United States (U.S. corporation)

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PATENT INFORMATION:
                       US 5403326 19950404
                                                                  <--
APPLICATION INFO.:
                       US 1993-12113 19930201 (8)
DOCUMENT TYPE:
                       Utility
PRIMARY EXAMINER:
                       Pellegrino, Stephen C.
ASSISTANT EXAMINER:
                       Schmidt, Jeffrey A.
LEGAL REPRESENTATIVE: Townsend & Townsend Khourie & Crew
NUMBER OF CLAIMS:
                       10
EXEMPLARY CLAIM:
NUMBER OF DRAWINGS:
                       16 Drawing Figure(s); 8 Drawing Page(s)
LINE COUNT:
                       540
      US 5403326 19950404
       . . . which will become the gastrostomy. This procedure with
      modifications is well known and is called a Percutaneous Endoscopic
      Gastrotomy or PEG.
DETD
       . . . is placed to gain access from the skin surface to the stomach
      lumen. Such ports are known and used for PEG procedures. See,
      for example, U.S. Pat. Nos. 4,863,438; 4,944,732; and 5,007,900 which
      are incorporated by reference herein. The port could.
       . . . bowel obstruction, a particularly dangerous complication of
DETD
      adhesions. A second advantage is most apparent in the high risk group
of
    neurologically damaged children. The minimally
      invasive nature of the inventive technique decreases surgical morbidity
      in this compromised population. Also performance of an. . .
L17 ANSWER 84 OF 117 USPATFULL
      Process for the preparation of 1,2,4-substituted imidazoles and related
       aminoalkylimidazole derivatives
ACCESSION NUMBER:
                       95:1749 USPATFULL
                       Process for the preparation of 1,2,4-substituted
TITLE:
                       imidazoles and related aminoalkylımidazole derivatives
                       McCort, Gary, Paris, France
INVENTOR(S):
                       Pascal, Jean-Claude, Cachan, France
PATENT ASSIGNEE(S):
                       Syntex Pharmaceuticals, Ltd., Maidenhead, England
                       (non-U.S. corporation)
                            NUMBER
                                         DATE
                       ______
PATENT INFORMATION: US 5378847 19950103
APPLICATION INFO.: US 1993-171594 19931221 (8)
RELATED APPLN. INFO.: Division of Ser. No. US 1992-46002, filed on 9 Apr
                       1992, now patented, Pat. No. US 5296609
DOCUMENT TYPE:
                       Utility
                      Brust, Joseph Paul
PRIMARY EXAMINER:
ASSISTANT EXAMINER:
                      Gabilan, Mary Susan H.
LEGAL REPRESENTATIVE: Lewis, Brian; Lowin, David A.; Krubiner, Alan M.
NUMBER OF CLAIMS:
                       10
EXEMPLARY CLAIM:
                       1
LINE COUNT:
                       1118
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       US 5378847 19950103
DETD
       . . . treated by direct neuronal protection, such as ischaemia
       including focal and global ischaemia, cerebral ischaemia including
       ischaemia-induced neurodegeneration, perinatal asphyxia, spinal
     injuries, peripheral nerve ischaemia, peripheral nerve
     damage, head trauma, primary intracerebral hemorrhage,
       encephalopathy, epilepsy or epileptic psychotic symptoms, and
       neurological diseases such as Alzheimer's, Huntington's chorea,
```

For systemic administration via suppository, traditional binders and

DETD

carriers include, for example, polyalkaline glycol or glycerides [e.g., PEG 1000 (96%) and PEG 4000 (4%)]. Such suppositories may be formed from mixtures containing active ingredients in the range of from about 0.5 wt/%. . . DETD . . . an index of ischemic damage insofar as an increase in binding of [.sup.3 H]-PK 11195 (assessed by B.sub.max) indirectly reflects neuronal damage. Compounds which prevent the increase in the number of binding sites are considered to be neuroprotective. L17 ANSWER 85 OF 117 USPATFULL Nucleic acid encoding neurotrophic factor four (NT-4), vectors, host cells and methods of production 94:99824 USPATFULL ACCESSION NUMBER: TITLE: Nucleic acid encoding neurotrophic factor four (NT-4), vectors, host cells and methods of production INVENTOR(S): Rosenthal, Arnon, Pacifica, CA, United States Genentech, Inc., South San Francisco, CA, United PATENT ASSIGNEE(S): (U.S. corporation) NUMBER DATE _____ PATENT INFORMATION: US 5364769 19941115 <--APPLICATION INFO.: US 1990-587707 19900925 (7) DOCUMENT TYPE: Utility PRIMARY EXAMINER: Hill, Jr., Robert J. PRIMARY EXAMINER: HILL, Jr., KODERU J. ASSISTANT EXAMINER: Allen, Marianne Porta LEGAL REPRESENTATIVE: Johnston, Sean A. NUMBER OF CLAIMS: EXEMPLARY CLAIM: 1 2 Drawing Figure(s); 2 Drawing Page(s) NUMBER OF DRAWINGS: 1357 LINE COUNT: CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5364769 19941115 DETD . . . loss of neurons, whether central, peripheral, or motorneurons. In addition, it may be useful for treating damaged nerve cells, e.g., nerves damaged by traumatic conditions such as burns and wounds, diabetes, kidney dysfunction, and the toxic effects of chemotherapeutics used to treat. sugar alcohols such as mannitol or sorbitol; salt-forming counterions such as sodium; and/or nonionic surfactants such as Tween, Pluronics or **PEG**. L17 ANSWER 86 OF 117 USPATFULL Retractor for spinal surgery 94:98904 USPATFULL ACCESSION NUMBER: TITLE: Retractor for spinal surgery Coker, Wesley L., 601 Enquirer Ave., Nashville, TN, INVENTOR(S): United States 37205 NUMBER DATE US 5363841 19941115 US 1993-86941 19930702 (8) PATENT INFORMATION: <--APPLICATION INFO.: DOCUMENT TYPE: Utility Apley, Richard J. PRIMARY EXAMINER: ASSISTANT EXAMINER: Maraglio, Donna L. LEGAL REPRESENTATIVE: Waddey, Jr., I. C. NUMBER OF CLAIMS: 16

EXEMPLARY CLAIM:

1

NUMBER OF DRAWINGS: 11 Drawing Figure(s); 7 Drawing Page(s)

LINE COUNT:

US 5363841 19941115 PΙ

SUMM . . be disastrous in that the screw can be misapplied, and tilting

either too far inward or outward can result in nerve

damage or ineffective stabilization of the spine.

The third distinction of this retractor over the prior art is SUMM laterally projecting anchor **peg** extending from the muscle side of the retractor blade which is meant to lie beneath the dorsolumbar fascia. This anchor peg further locks the retractor into the

depths of the wound and-prevents its migration up and out of the wound.

L17 ANSWER 87 OF 117 USPATFULL

Glaucoma treatment

94:79972 USPATFULL ACCESSION NUMBER: TITLE: Glaucoma treatment

INVENTOR(S): Stein, Herman H., Highland Park, IL, United States

Plattner, Jacob J., Libertyville, IL, United States

Crowley, Steven R., Vernon Hills, IL, United States Abbott Laboratories, Abbott Park, IL, United States PATENT ASSIGNEE(S):

(U.S. corporation)

NUMBER DATE -----

PATENT INFORMATION: US 5346887 19940913 <---

APPLICATION INFO.: US 1991-690148 19910423 (7)

RELATED APPLN. INFO.: Division of Ser. No. US 1990-488810, filed on 2 Mar

1990, now patented, Pat. No. US 5036051 which is a division of Ser. No. US 1988-240567, filed on 8 Sep 1988, now patented, Pat. No. US 4927807 which is a continuation-in-part of Ser. No. US 1987-105636, filed

on 6 Oct 1987, now abandoned

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Fay, Zohreh A. LEGAL REPRESENTATIVE: Crowley, Steven R.

NUMBER OF CLAIMS: EXEMPLARY CLAIM: 1 LINE COUNT: 2977

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5346887 19940913

SUMM Glaucoma is a condition characterized by an increase in intraocular pressure. Increased intraocular pressure can lead to optic nerve damage and defects in the visual field. Blindness can result if

the condition is left untreated.

SUMM . . . such as dextran, hydroxyloweralkyl dextran, carboxymethyl dextran, hydroxyloweralkyl cellulose, loweralkyl cellulose, carboxymethyl cellulose, polyvinyl alcohol, dextrin, starch, polyvinyl pyrrolidone and polyalkylene glycols may be used as the carrier for the drug.

L17 ANSWER 88 OF 117 USPATFULL

Demineralized bone particles and flowable osteogenic composition containing same

ACCESSION NUMBER: 94:44221 USPATFULL

TITLE: Demineralized bone particles and flowable osteogenic

composition containing same

INVENTOR(S): Prewett, Annamarie B., Little Silver, NJ, United

States

Stikeleather, Roger C., Doylestown, PA, United States

PATENT ASSIGNEE(S): Osteotech, Inc., Shrewsbury, NJ, United States (U.S.

corporation)

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NUMBER
                                        DATE
PATENT INFORMATION: US 5314476 19940524 APPLICATION INFO.: US 1993-119882 19930910 (8)
                                                                  <--
RELATED APPLN. INFO.: Continuation of Ser. No. US 1992-830934, filed on 4
                       1992, now abandoned
                  Utility
DOCUMENT TYPE:
PRIMARY EXAMINER:
                      Isabella, David
ASSISTANT EXAMINER:
                      Nguyen, Dinh X.
LEGAL REPRESENTATIVE: Dilworth & Barrese
NUMBER OF CLAIMS:
                       20
EXEMPLARY CLAIM:
LINE COUNT:
                       481
      US 5314476 19940524
       . . . derivatives of the foregoing. Specific polyhydroxy compounds
DETD
       include ethylene glycol, diethylene glycol, triethylene glycol,
       1,2-propanediol, glycerol, trimethylolethane, trimethylolpropane,
       erythritol, pentaerythritol, polyalkylene glycols
       such as the polyethylene glycols, xylitol, sorbitol, mannitol,
dulcitol,
       arabinose, xylose, ribose, adonitol, arabitol, rhamose, inositol,
       fructose, galactose, glucose, mannose,. .
       . . fixation, tumor surgery, e.g., deficit filling, discectomy,
DETD
       laminectomy, excision of spinal cord tumors, anterior cervical and
       thoracic operations, repair of spinal injuries,
       scoliosis, lordosis and kyphosis treatments, intermaxillary fixation of
       fractures, mentoplasty, temporomandibular joint replacement, alveolar
       ridge augmentation and reconstruction, inlay bone. . .
L17 ANSWER 89 OF 117 USPATFULL
      Tank for electroanesthetizing fish
                      94:34591 USPATFULL
ACCESSION NUMBER:
TITLE:
                       Tank for electroanesthetizing fish
                       Sharber, Norman G., 515 W. Havasupi Rd., Flagstaff,
INVENTOR(S):
AZ,
                       United States 86001
                            NUMBER
                       -----
                      US 5305711 19940426
PATENT INFORMATION:
                                                                   <--
                    US 1993-17384 19930212 (8)
APPLICATION INFO.:
RELATED APPLN. INFO.: Continuation of Ser. No. US 1992-874715, filed on 27
                       Apr 1992, now patented, Pat. No. US 5253610
DOCUMENT TYPE:
                       Utility
PRIMARY EXAMINER:
                      Swiatek, Robert P.
LEGAL REPRESENTATIVE: Cahill, Sutton & Thomas
NUMBER OF CLAIMS:
                       20
EXEMPLARY CLAIM:
NUMBER OF DRAWINGS:
                      9 Drawing Figure(s); 3 Drawing Page(s)
LINE COUNT:
                       411
       US 5305711 19940426
       . . . fish. As will be described below, use of tank 10 produces
DETD
petit
       mal in fish without the muscle, bone and spinal
     injuries resulting from presently used apparatus for inducing
       electroanesthesia. Furthermore, use of tank 10 eliminates the need for
       chemicals to render. . .
```

. . . of the fish to be placed within the tank. Such adjustment may

be readily performed by providing a plurality of **pegs** 58,60

DETD

defining a number of columns in each of diffuser plates 22,20 for receiving and maintaining moveable wall 54. That is, the moveable wall may be placed between adjacent pairs of **pegs** toward or away from fixed wall 56 to temporarily set the width of the portion of the tank between movable. . .

L17 ANSWER 90 OF 117 USPATFULL

TI Process for the preparation of 1,2,4-substituted imidazoles and related

aminoalkylimidazole derivatives

ACCESSION NUMBER: 94:24441 USPATFULL

TITLE: Process for the preparation of 1,2,4-substituted

imidazoles and related aminoalkylimidazole derivatives

INVENTOR(S): McCort, Gary, Paris, France

Pascal, Jean-Claude, Cachan, France

PATENT ASSIGNEE(S): Syntex Pharmaceuticals, Ltd., Maidenhead, United

Kingdom (non-U.S. corporation)

NUMBER DATE

PATENT INFORMATION: US 5296609 19940322 <--

APPLICATION INFO.: US 1993-46002 19930409 (8)

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Lee, Mary C.
ASSISTANT EXAMINER: McKane, Joseph K.

LEGAL REPRESENTATIVE: Lewis, Brian; Lowin, David A.; Krubiner, Alan M.

NUMBER OF CLAIMS: 20 EXEMPLARY CLAIM: 1 LINE COUNT: 1110

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 5296609 19940322

SUMM . . . treated by direct neuronal protection, such as ischaemia including focal and global ischaemia, cerebral ischaemia including

ischaemia-induced neurodegeneration, perinatal asphyxia, spinal

injuries, peripheral nerve ischaemia, peripheral nerve

damage, head trauma, primary intracerebral hemorrhage, encephalopathy, epilepsy or epileptic psychotic symptoms, and neurological diseases such as Alzheimer's, Huntington's chorea,

Parkinsons.

SUMM . . . treated by direct neuronal protection, such as ischaemia including focal and global ischaemia, cerebral ischaemia including ischaemia-induced neurodegeneration, perinatal asphyxia, **spinal**

injuries, peripheral nerve ischaemia, peripheral nerve damage, head trauma, primary intracerebral hemorrhage,

encephalopathy, epilepsy or epileptic psychotic symptoms, and neurological diseases such as Alzheimer's, Huntington's chorea,

Parkinsons. .

SUMM For systemic administration via suppository, traditional binders and carriers include, for example, polyalkaline glycol or glycerides [e.g.,

PEG 1000 (96%) and **PEG** 4000 (4%)]. Such suppositories

may be formed from mixtures containing active ingredients in the range of from about 0.5 wt/%. . .

L17 ANSWER 91 OF 117 USPATFULL

TI Substituted imidazolyl-alkyl-piperazine and -diazepine derivatives

ACCESSION NUMBER: 94:1427 USPATFULL

TITLE: Substituted imidazolyl-alkyl-piperazine and -diazepine

derivatives

INVENTOR(S): Pascal, Jean C., Cachan, France

Lee, Chi-Ho, Palo Alto, CA, United States

Alps, Brian J., Linlithgow, Scotland

Pinhas, Henri, Paris, France

Whiting, Roger L., Los Altos, CA, United States MacFarlane, Calum B., Linlithgow, Scotland Beranger, Serge, Bretigny-sur-Cedres, France

Dow, Robert J., Edinburgh, Scotland

Syntex Pharmaceutical Ltd., Maidenhead, England PATENT ASSIGNEE(S):

(non-U.S. corporation)

NUMBER DATE _____

PATENT INFORMATION:

APPLICATION INFO.:

US 5276034 19940104 <-US 1991-688193 19910419 (7)
Division of Ser. No. US 1988-260969, filed on 21 Oct RELATED APPLN. INFO.: 1988, now patented, Pat. No. US 5043447 which is a continuation-in-part of Ser. No. US 1987-42181, filed

on 24 Apr 1987, now patented, Pat. No. US 4829065

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Tsang, Cecilia

LEGAL REPRESENTATIVE: Lowin, David A.; Moran, Tom M.; Desjardins, Cathleen

16 NUMBER OF CLAIMS: EXEMPLARY CLAIM: 1 2218 LINE COUNT:

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5276034 19940104 diseases treated by direct neuronal protection, such as ischemia PARN

including focal and global ischemia, spinal injuries , head trauma, and neurological diseases such as Alzheimer's and Huntington's chorea;

diseases treated by direct neuronal protection, such as ischemia PARN including focal and global ischemia, spinal injuries , head trauma, and neurological diseases such as Alzheimer's and Huntington's chorea;

diseases treated by direct neuronal protection, such as ischemia PARN including focal and global ischemia, spinal injuries , head trauma, and neurological diseases such as Alzheimer's and Huntington's chorea;

For systemic administration via suppository, traditional binders and PARN carriers include, for example, polyalkaline glycol or triglycerides [e.g., PEG 1000 (96% and PEG 4000 (4%)]. Such suppositories may be formed from mixtures containing active ingredients in the range of from about 0.5 wt/%. . .

L17 ANSWER 92 OF 117 USPATFULL

Screening method for neuroprotective compounds

93:98315 USPATFULL ACCESSION NUMBER:

Screening method for neuroprotective compounds TTTLE:

Miljanich, George P., Redwood City, CA, United States INVENTOR(S): Bitner, Robert S., Mountain View, CA, United States

Bowersox, Stephen S., Menlo Park, CA, United States Fox, James A., Palo Alto, CA, United States

Valentino, Karen L., San Carlos, CA, United States Yamashiro, Donald H., San Francisco, CA, United States Tsubokawa, Makoto, South San Francisco, CA, United

States

Neurex Corporation, Menlo Park, CA, United States PATENT ASSIGNEE(S):

(U.S.

corporation)

NUMBER DATE

US 5264371 19931123 <--PATENT INFORMATION:

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19920323 (7)
APPLICATION INFO.:
                        US 1992-855269
                        Division of Ser. No. US 1990-561766, filed on 2 Aug
RELATED APPLN. INFO.:
                        1990, now patented, Pat. No. US 5189020 which is a
                        continuation-in-part of Ser. No. US 1989-440094, filed
                        on 22 Nov 1989, now patented, Pat. No. US 5051403
DOCUMENT TYPE:
                        Utility
                        Russel, Jeffrey E.
PRIMARY EXAMINER:
                        Stratford, Carol A.; Dehlinger, Peter J.
LEGAL REPRESENTATIVE:
NUMBER OF CLAIMS:
EXEMPLARY CLAIM:
                        22 Drawing Figure(s); 12 Drawing Page(s)
NUMBER OF DRAWINGS:
LINE COUNT:
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       US 5264371 19931123
                                                                     <--
DETD
       Co-owned U.S. patent application for "Method of Treating
       Ischemia-Related Neuronal Damage," Ser. No. 440,094 filed Nov. 22, 1989, now U.S. Pat. No. 5,051,403, describes a method of
       reducing neuronal damage related to ischemia, by
       administering OCT peptides which have certain binding and/or inhibitory
       properties. The properties which were found to.
DETD
       In vitro and in vivo studies reported in the above-cited patent
       application for "Method of Treating Ischemia-Related Neuronal
    Damage, " demonstrate a strong correlation between (a) high
       binding affinity to synaptosomal membranes, (b) inhibition of
       voltage-gated calcium ion currents and neurotransmitter release
       selectively in neuronal cells, and (c) ability to reduce
    neuronal damage in ischemia-related injury, such as
       stroke. The mechanism of neural protection by high-affinity OCT
peptides
       presumably involves inhibition of voltage-gated. . . and the
       consequent release of neurotransmitters from the cells. This mechanism
       of OCT protection is consistent with the finding that neuronal
     damage in ischemia-related injury is associated with elevated
       intracellular calcium levels (Deshpande et al.).
DETD
       . . . effective inhibitors of voltage-gated calcium currents in
       neuronal cells, and that such compounds, in turn, are useful for
       reducing ischemia-related neuronal damage, such as
       caused by stroke. This model is the basis of the screening method of
the
DETD
       . . . the invention facilitates the screening of effective
       neuroprotective compounds. One criterion for an effective
       neuroprotective compound, for use in reducing neuronal
     damage in ischemia-related injury, is the ability to inhibit the
       spread of neuronal damage from the site of injury.
       Evidence indicates that the spread of damage in ischemia-related injury
       is due, at least in. . .
DETD
       In another aspect, the present invention provides a treatment method
for
       reducing neuronal damage related to an ischemic
       condition in a human patient, by administration of a pharmaceutically
       effective amount of a compound selected.
       . . to OCT binding sites in neuronal tissue and (b) selective
DETD
       inhibition of calcium channel currents and neurotransmitter release in
       reducing neuronal damage in ischemia-related injury.
       Based on the apparent mechanism of action of the OCT peptides, it can
be
       predicted that screened.
DETD
       . . . through 0.6% polyethyleneimine treated GF/C filters
(Millipore)
       on a Millipore filtration unit. Protein bound [.sup.125 I]MVIIA OCT
```

present in the **PEG** precipitate was determined by gamma counting. FIG. 7 illustrates displacement of [.sup.125 I]-MVIIA OCT binding by unlabeled MVIIA OCT in. . .

L17 ANSWER 93 OF 117 USPATFULL

TI Tank for electroanesthetizing fish ACCESSION NUMBER: 93:86402 USPATFULL

TITLE: Tank for electroanesthetizing fish

INVENTOR(S): Sharber, Norman G., 515 W. Havasupi Rd., Flagstaff,

AZ,

United States 86001

NUMBER DATE

PATENT INFORMATION: US 5253610 19931019 <-- APPLICATION INFO.: US 1992-874715 19920427 (7)

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Swiatek, Robert P. LEGAL REPRESENTATIVE: Cahill, Sutton & Thomas

NUMBER OF CLAIMS: 24 EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 9 Drawing Figure(s); 3 Drawing Page

LINE COUNT: 413 PI US 5253610 19931019

DETD . . . fish. As will be described below, use of tank 10 produces

petit

mal in fish without the muscle, bone and spinal,

injuries resulting from presently used apparatus for inducing
 electroanesthesia. Furthermore, use of tank 10 eliminates the need for
 chemicals to render. . .

DETD . . . of the fish to be placed within the tank. Such adjustment may be readily performed by providing a plurality of **pegs** 58,60 defining a number of columns in each of diffuser plates 22,20 for receiving and maintaining moveable wall 54. That is, the moveable wall may be placed between adjacent pairs of **pegs** toward or away from fixed wall 56 to temporarily set the width of the portion of the tank between movable. . .

L17 ANSWER 94 OF 117 USPATFULL

TI Substituted imidazolyl-alkyl-piperazine and -diazepine derivatives

ACCESSION NUMBER: 93:85286 USPATFULL

TITLE: Substituted imidazolyl-alkyl-piperazine and -diazepine

derivatives

INVENTOR(S): Pascal, Jean C., Cachan, France

Lee, Chi-Ho, Palo Alto, CA, United States Alps, Brian J., Linlithgow, Scotland

Pinhas, Henri, Paris, France

Whiting, Roger L., Los Altos, CA, United States Syntex Pharmaceuticals, Ltd., Maidenhead, England

<--

(non-U.S. corporation)

NUMBER DATE

TENT INFORMATION: US 5252736 19931012

PATENT INFORMATION: US 5252736 19931012 APPLICATION INFO.: US 1991-789230 19911107 (7)

DISCLAIMER DATE: 20090225

PATENT ASSIGNEE(S):

RELATED APPLN. INFO.: Continuation of Ser. No. US 1991-652141, filed on 7

Feb

1991, now patented, Pat. No. US 5091428 which is a division of Ser. No. US 1990-505379, filed on 6 Apr 1990, now patented, Pat. No. US 5010075 which is a

division of Ser. No. US 1989-313656, filed on 21 Feb 1989, now patented, Pat. No. US 4935417 which is a division of Ser. No. US 1987-42181, filed on 24 Apr

1987, now patented, Pat. No. US 4829065

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Tsang, Cecilia

LEGAL REPRESENTATIVE: Desjardins, Cathleen; Lowin, David A.; Moran, Tom M.

NUMBER OF CLAIMS: 7
EXEMPLARY CLAIM: 1
LINE COUNT: 1436

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 5252736 19931012 <--

SUMM . . . a variety of disease states, such as stroke, epilepsy, hypertension, angina, migraine, arrhythmia, thrombosis, embolism and also for treatment of **spinal injuries**.

SUMM . . . a variety of disease states, such as stroke, epilepsy, hypertension, angina, migraine, arrhythmia, thrombosis, embolism and also for treatment of **spinal injuries**, comprising administering a therapeutically effective amount of compound of Formula A to a mammal.

SUMM . . . include stroke, migraine, epilepsy, hypertension, angina, arrhythmia, thrombosis, and embolism. The compounds of this invention are also useful for treating **spinal injuries**, and are particularly useful for treating cerebrovascular disease states,

for

example, stroke.

SUMM For systemic administration via suppository, traditional binders and carriers include, for example, polyalkaline glycol or triglycerides [e.g., PEG 1000 (96%) and PEG 4000 (4%)]. Such suppositories may be formed from mixtures containing active ingredients in the range of from about 0.5 wt/%. . .

L17 ANSWER 95 OF 117 USPATFULL

TI Method of reducing neuronal damage using omega conotoxin peptides

ACCESSION NUMBER: 93:14551 USPATFULL

TITLE: Method of reducing neuronal damage using omega

conotoxin peptides

INVENTOR(S): Miljanich, George P., Redwood City, CA, United States

Bitner, Robert S., Mountain View, CA, United States Bowersox, Stephen S., Menlo Park, CA, United States

Fox, James A., Palo Alto, CA, United States

Valentino, Karen L., San Carlos, CA, United States Yamashiro, Donald H., San Francisco, CA, United States Tsubokawa, Makoto, South San Francisco, CA, United

<--

States

PATENT ASSIGNEE(S): Neurex Corporation, Menlo Park, CA, United States

(U.S.

corporation)

PATENT INFORMATION: US 5189020 19930223 APPLICATION INFO.: US 1990-561766 19900802 (7)

DISCLAIMER DATE: 20080924

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1989-440094, filed

on 22 Nov 1989, now patented, Pat. No. US 5051403

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Cashion, Jr., Merrell C.

ASSISTANT EXAMINER: Rozycki, Andrew G. LEGAL REPRESENTATIVE: Dehlinger, Peter J.

NUMBER OF CLAIMS:

EXEMPLARY CLAIM: 22 Drawing Figure(s); 12 Drawing Page(s) NUMBER OF DRAWINGS: LINE COUNT: 1895 CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5189020 19930223 . . Treating Ischemia-Related Neuronal Ser. No. 440,094 filed Nov. DETD 22, 1989, now U.S. Pat. No. 5,051,403, describes a method of reducing neuronal damage related to ischemia, by administering OCT peptides which have certain binding and/or inhibitory properties. The properties which were found to. In vitro and in vivo studies reported in the above-cited patent DETD application for "Method of Treating Ischemia-Related Neuronal Damage," demonstrate a strong correlation between (a) high binding affinity to synaptosomal membranes, (b) inhibition of voltage-gated calcium ion currents and neurotransmitter release selectively in neuronal cells, and (c) ability to reduce neuronal damage in ischemia-related injury, such as stroke. The mechanism of neural protection by high-affinity OCT peptides presumably involves inhibition of voltage-gated. . . and the consequent release of neurotransmitters from the cells. This mechanism of OCT protection is consistent with the finding that ${\tt neuronal}$ damage in ischemia-related injury is associated with elevated intracellular calcium levels (Deshpande et al.). DETD . . . effective inhibitors of voltage-gated calcium currents in neuronal cells, and that such compounds, in turn, are useful for reducing ischemia-related neuronal damage, such as caused by stroke. This model is the basis of the screening method of the invention. DETD . . . the invention facilitates the screening of effective neuroprotective compounds. One criterion for an effective neuroprotective compound, for use in reducing neuronal damage in ischemia-related injury, is the ability to inhibit the spread of neuronal damage from the site of injury. Evidence indicates that the spread of damage in ischemia-related injury is due, at least in. DETD In another aspect, the present invention provides a treatment method for reducing neuronal damage related to an ischemic condition in a human patient, by administration of a pharmaceutically effective amount of a compound selected. DETD . . to OCT binding sites in neuronal tissue and (b) selective inhibition of calcium channel currents and neurotransmitter release in reducing neuronal damage in ischemia-related injury. Based on the apparent mechanism of action of the OCT peptides, it can be predicted that screened. . . . through 0.6% polyethyleneimine treated GF/C filters DETD (Millipore) on a Millipore filtration unit. Protein bound [.sup.125 I]MVIIA OCT present in the PEG precipitate was determined by gamma counting. FIG. 7 illustrates displacement of [.sup.125 I]-MVIIA OCT binding by unlabeled MVIIA OCT in. ANSWER 96 OF 117 USPATFULL T.17 TΙ Stoma creator gastrostomy device and method for placement of a feeding tube

92:98608 USPATFULL

placement of a feeding tube

Stoma creator gastrostomy device and method for

ACCESSION NUMBER:

TITLE:

Isaac, Ronald M., Libertyville, IL, United States Hirsch, William H., Columbus, OH, United States Abbott Laboratories, Abbott Park, IL, United States PATENT ASSIGNEE(S): (U.S. corporation) NUMBER DATE US 5167627 19921201 US 1991-701914 19910517 (7) PATENT INFORMATION: APPLICATION INFO.: RELATED APPLN. INFO.: Division of Ser. No. US 1990-581952, filed on 13 Sep 1990 DOCUMENT TYPE: Utility PRIMARY EXAMINER: Jaworski, Francis Akers, Scott R. ASSISTANT EXAMINER: LEGAL REPRESENTATIVE: Drayer, Lonnie R.; Nickey, Donald O. NUMBER OF CLAIMS: 10 EXEMPLARY CLAIM: NUMBER OF DRAWINGS: 12 Drawing Figure(s); 5 Drawing Page(s) LINE COUNT: 457 US 5167627 19921201 . . . unable to ingest enough solid food to meet their body's nutritional needs. Examples of these individuals would include stroke or neurologically impaired patients, who have lost their ability to swallow effectively; critically ill, weak or comatose patients, who may be unable to. surgical procedure utilizing a general or local anesthetic, SUMM the preferred method for placement of these ports is percutaneous endoscopic gastrotomy (PEG) that involves use of an endoscope to visualize the insertion site on the gastric mucosa and the subsequent creation of. . . SUMM Yet another aspect of this invention is that it is less time consuming than some of the other PEG procedures. L17 ANSWER 97 OF 117 USPATFULL Pelvic belt with hand mounts for spinal unloading ACCESSION NUMBER: 92:88212 USPATFULL TITLE: Pelvic belt with hand mounts for spinal unloading INVENTOR(S): Jalalian, Armen, 76 Hernandez Ave., San Francisco, CA, United States 94127 NUMBER DATE US 5158098 19921027 PATENT INFORMATION: US 1992-821278 19920110 (7) APPLICATION INFO.: Continuation of Ser. No. US 1990-588443, filed on 26 RELATED APPLN. INFO.: Sep 1990, now abandoned DOCUMENT TYPE: Utility

INVENTOR(S):

PRIMARY EXAMINER:

Clegg, Robert D., Pickerington, OH, United States

Hanlon, Brian E. ASSISTANT EXAMINER: LEGAL REPRESENTATIVE: Flehr, Hohbach, Test, Albritton & Herbert NUMBER OF CLAIMS: 30 EXEMPLARY CLAIM: NUMBER OF DRAWINGS: 4 Drawing Figure(s); 1 Drawing Page(s) LINE COUNT: 317 PΙ US 5158098 19921027 . . . forces of the upper body, head and arms, orthotic appliances SUMM such as belts, girdles, corsets and braces aid in preventing

Hafer, Robert A.

spinal injury and are used during therapy to support

the spinal column. Such orthotic appliances unload the spine,

immobilize

the spine, or.

SUMM

. . . of the wearer's hand is flush with the wearer's back. Alternatively, the hand grips may be a handle or a peg which extends out substantially perpendicular to the waistband. The wearer

grips onto the hand grip and pushes downward.

L17 ANSWER 98 OF 117 USPATFULL

Nerve growth factor peptides

ACCESSION NUMBER:

92:61895 USPATFULL

TITLE:

Nerve growth factor peptides

INVENTOR(S):

Mobley, William C., Moraga, CA, United States

Longo, Frank M., San Francisco, CA, United States Kauer, James C., Kennett Square, PA, United States

PATENT ASSIGNEE(S):

Regents of the University of California, Berkeley, CA,

United States (U.S. corporation)

DATE NUMBER

PATENT INFORMATION:

US 5134121 19920728

APPLICATION INFO.:

US 1991-640577 19910114 (7)

RELATED APPLN. INFO.:

Continuation of Ser. No. US 1989-299698, filed on 23

Jan 1989, now abandoned which is a

continuation-in-part

of Ser. No. US 1988-173975, filed on 28 Mar 1988, now

abandoned

DOCUMENT TYPE:

Utility

PRIMARY EXAMINER:

Cashion, Jr., Merrell C.

ASSISTANT EXAMINER:

Perkins, Susan M.

NUMBER OF CLAIMS:

EXEMPLARY CLAIM:

8 Drawing Figure(s); 7 Drawing Page(s)

NUMBER OF DRAWINGS: LINE COUNT:

1133

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5134121 19920728

Analogs of NGF fragments with NGF activity as described above have potentialpharmaceutical applications in situations involving

nerve damage from traumatic accidents, stroke and encephalitis.

DETD

. . . excipients include water, saline, Ringer's solution, dextrose solution, and solutions of ethanol, glucose, sucrose, dextran, mannose,

mannitol, sorbitol, polyethylene glycol (PEG), phosphate,

acetate, gelatin, collagen, and the like. One may additionally include suitable preservatives, stabilizers, antioxidants, antimicrobials, buffering agents and the.

L17 ANSWER 99 OF 117 USPATFULL

Substituted imidazolyl-alkyl-piperazine and -diazepine derivatives

92:15054 USPATFULL ACCESSION NUMBER:

Substituted imidazolyl-alkyl-piperazine and -diazepine TITLE:

derivatives

INVENTOR(S): Pascal, Jean C., Cachan, France

Lee, Chi-Ho, Palo Alto, CA, United States

Alps, Brian J., Linlithgow, Scotland

Pinhas, Henri, Paris, France

Whiting, Roger L., Los Altos, CA, United States

Syntex Pharmaceuticals, Ltd., Hamilton, Bermuda PATENT ASSIGNEE(S):

(non-U.S. corporation)

NUMBER DATE _____ US 5091428 19920225 PATENT INFORMATION: US 1991-652141 19910207 (7) Division of Ser. No. US 1990-505379, filed on 6 Apr APPLICATION INFO.: RELATED APPLN. INFO.: 1990, now patented, Pat. No. US 5010075 which is a division of Ser. No. US 1989-313656, filed on 21 Feb 1989, now patented, Pat. No. US 4938417 which is a division of Ser. No. US 1987-42181, filed on 24 Apr 1987, now patented, Pat. No. US 4829065 DOCUMENT TYPE: Utility PRIMARY EXAMINER: Shen, Cecilia LEGAL REPRESENTATIVE: Lowin, David A.; Moran, Tom M. NUMBER OF CLAIMS: 31 EXEMPLARY CLAIM: LINE COUNT: 1486 CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5091428 19920225 . . . a variety of disease states, such as stroke, epilepsy, SUMM hypertension, angina, migraine, arrhythmia, thrombosis, embolism and also for treatment of spinal injuries. . . . a variety of disease states, such as stroke, epilepsy, SUMM hypertension, angina, migraine, arrhythmia, thrombosis, embolism and also for treatment of spinal injuries, comprising administering a therapeutically effective amount of compound of Formula A to a mammal. . . . include stroke, migraine, epilepsy, hypertension, angina, SUMM arrhythmia, thrombosis, and embolism. The compounds of this invention are also useful for treating spinal injuries, and are particularly useful for treating cerebrovascular disease states, for example, stroke. For systemic administration via suppository, traditional binders and SUMM carriers include, for example, polyalkaline glycol or triglycerides [e.g., PEG 1000 (96%) and PEG 4000 (4%)]. Such suppositories may be formed from mixtures containing active ingredients in the range of from about 0.5 wt/%. . L17 ANSWER 100 OF 117 USPATFULL Stoma creator gastrostomy device and method for placement of a feeding tube ACCESSION NUMBER: 91:103725 USPATFULL Stoma creator gastrostomy device and method for TITLE: placement of a feeding tube Clegg, Robert D., Pickerington, OH, United States INVENTOR(S): Isaac, Ronald M., Libertyville, IL, United States Hirsch, William H., Columbus, OH, United States Abbott Laboratories, Abbott Park, IL, United States PATENT ASSIGNEE(S): (U.S. corporation) NUMBER DATE US 5074846 19911224 PATENT INFORMATION: <--US 1990-581952 19900913 (7) APPLICATION INFO.: DOCUMENT TYPE: Utility PRIMARY EXAMINER: Jaworski, Francis ASSISTANT EXAMINER: Akers, Scott R.

Drayer, Lonnie R.; Nickey, Donald O.

12 Drawing Figure(s); 5 Drawing Page(s)

LEGAL REPRESENTATIVE: NUMBER OF CLAIMS:

EXEMPLARY CLAIM: NUMBER OF DRAWINGS: 3

LINE COUNT: 424 US 5074846 19911224 PΙ . . . unable to ingest enough solid food to meet their body's SUMM nutritional needs. Examples of these individuals would include stroke or neurologically impaired patients, who have lost their ability to swallow effectively; critically ill, weak or comatose patients, who may be unable to. surgical procedure utilizing a general or local anesthetic, SUMM the preferred method for placement of these ports is percutaneous endoscopic gastrotomy (PEG) that involves us of an endoscope to visualize the insertion site on the gastric mucosa and the subsequent creation of. . . Yet another aspect of this invention is that it is less time consuming SUMM than some of the other PEG procedures. L17 ANSWER 101 OF 117 USPATFULL Parenteral formulations of 1-diphenylmethyl-4-((2-(4-methylphenyl)-5methyl-1H-imidazol-4-yl)methyl)piperazine ACCESSION NUMBER: 91:90754 USPATFULL Parenteral formulations of 1-diphenylmethyl-4-((2-(4-TITLE: methylphenyl)-5-methyl-1H-imidazol-4yl)methyl)piperazine Selkirk, Alastair B., Edinburgh, Scotland INVENTOR(S): Dey, Michael J., West Lothian, Scotland Syntex Pharmaceuticals, Ltd., Maidenhead, England PATENT ASSIGNEE(S): (non-U.S. corporation) DATE NUMBER _____ US 5063220 19911105 <--PATENT INFORMATION: US 1990-585436 19900920 (7) APPLICATION INFO.: Division of Ser. No. US 1988-260628, filed on 21 Oct RELATED APPLN. INFO.: 1988, now patented, Pat. No. US 4973591 DOCUMENT TYPE: Utility PRIMARY EXAMINER: Waddell, Frederick E. ASSISTANT EXAMINER: Fay, Zohreh A. LEGAL REPRESENTATIVE: Lowin, David A.; Moran, Tom M. NUMBER OF CLAIMS: 9 EXEMPLARY CLAIM: 434 LINE COUNT: CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5063220 19911105 PΙ diseases treated by direct neuronal protection, such as ischemia SUMM including focal and global ischemia, spinal injuries , head trauma, and neurological diseases such as Alzheimer's and Huntington's chorea; . . . of aqueous miscible cosolvents commonly used to improve the SUMM solubility of parenteral products [for example, various mixtures of polyethylene glycol (PEG 300), propylene glycol (PG) and ethanol], achieved limited increase in the solubility of the active agent. However, all of the. . . diseases treated by direct neuronal protection, such as ischemia SUMM including focal and global ischemia, spinal injuries , head trauma, and neurological diseases such as Alzheimer's and Huntington's chorea;

L17 ANSWER 102 OF 117 USPATFULL

Glaucoma treatment

TΙ

ACCESSION NUMBER:

TITLE:

91:86729 USPATFULL Glaucoma treatment

INVENTOR(S):

Stein, Herman H., Highland Park, IL, United States Plattner, Jacob J., Libertyville, IL, United States Crowley, Steven R., Vernon Hills, IL, United States Abbott Laboratories, Abbott Park, IL, United States

PATENT ASSIGNEE(S):

(U.S. corporation)

NUMBER DATE NOMBER DATE

PATENT INFORMATION: US 5059589 19911022

APPLICATION INFO.:

RELATED APPLN. INFO.:

US 1990-488572 19900302 (7) Division of Ser. No. US 1988-240567, filed on 8 Sep 1988, now patented, Pat. No. US 4927807 which is a continuation-in-part of Ser. No. US 1987-105636, filed

on 6 Oct 1987, now abandoned

DOCUMENT TYPE:

Utility

PRIMARY EXAMINER:

Waddell, Frederick E.

ASSISTANT EXAMINER:

Fay, Zohreh A.

LEGAL REPRESENTATIVE: Crowley, Steven R.; Weinstock, Steven F.

NUMBER OF CLAIMS: EXEMPLARY CLAIM:

1

LINE COUNT:

3143

CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 5059589 19911022

SUMM

Glaucoma is a condition characterized by an increase in intraocular pressure. Increased intraocular pressure can lead to optic nerve

damage and defects in the visual field. Blindness can result if

the condition is left untreated.

SUMM

. . . such as dextran, hydroxyloweralkyl dextran, carboxymethyl dextran, hydroxyloweralkyl cellulose, loweralkyl cellulose, carboxymethyl cellulose, polyvinyl alcohol, dextrin, starch, polyvinyl pyrrolidone and polyalkylene glycols may be used as

the carrier for the drug.

L17 ANSWER 103 OF 117 USPATFULL

Substituted imidazolyl-alkyl-piperazine and -diazepine derivatives

ACCESSION NUMBER:

91:68999 USPATFULL

TITLE:

Substituted imidazolyl-alkyl-piperazine and -diazepine

derivatives

INVENTOR(S):

Pascal, Jean C., Cachan, France

Lee, Chi-Ho, Palo Alto, CA, United States

Alps, Brian J., Linlithgow, Scotland

Pinhas, Henri, Paris, France

Whiting, Roger L., Los Altos, CA, United States Macfarlane, Calum B., Linlithgow, Scotland Beranger, Serge, Bretigny-Sur-Cedres, France

Dow, Robert J., Edinburgh, Scotland

PATENT ASSIGNEE(S):

Syntex Pharmaceuticals, Ltd., Berkshire, England

(non-U.S. corporation)

NUMBER

PATENT INFORMATION:

US 5043447 19910827

APPLICATION INFO.:

US 1988-260969 19881021 (7)

RELATED APPLN. INFO.:

Continuation-in-part of Ser. No. US 1987-42181, filed

on 24 Apr 1987, now patented, Pat. No. US 4829065

NUMBER DATE

PRIORITY INFORMATION: EP 1988-303646 19880422

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Shen, Cecilia

LEGAL REPRESENTATIVE: Lowin, David A.; Moran, Tom M.

NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
LINE COUNT: 2177

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 5043447 19910827 <-- PARN diseases treated by direct neuronal protection, such as ischemia

PARN diseases treated by direct neuronal protection, such as ischemia including focal and global ischemia, **spinal injuries**

, head trauma, and neurological diseases such as Alzheimer's and Huntington's chorea;

PARN diseases treated by direct neuronal protection, such as ischemia including focal and global ischemia, **spinal injuries**, head trauma, and neurological diseases such as Alzheimer's and

Huntington's chorea;

PARN diseases treated by direct neuronal protection, such as ischemia including focal and global ischemia, **spinal injuries**, head trauma, and neurological diseases such as Alzheimer's and Huntington's chorea;

PARN For systemic administration via suppository, traditional binders and carriers include, for example, polyalkaline glycol or triglycerides [e.g., PEG 1000 (96%) and PEG 4000 (4%)]. Such suppositories may be formed from mixtures containing active ingredients in the range of from about 0.5 wt/%. . .

L17 ANSWER 104 OF 117 USPATFULL

TI Glaucoma treatment

ACCESSION NUMBER: 91:60794 USPATFULL TITLE: Glaucoma treatment

INVENTOR(S): Stein, Herman H., Highland Park, IL, United States Plattner, Jacob J., Libertyville, IL, United States

PATENT ASSIGNEE(S): Abbott Laboratories, Abbott Park, IL, United States

(U.S. corporation)

NUMBER DATE

PATENT INFORMATION: US 5036051 19910730 APPLICATION INFO.: US 1990-488810 19900302 (7)

RELATED APPLN. INFO.: Division of Ser. No. US 1988-240567, filed on 8 Sep

1988, now patented, Pat. No. US 4927807

Continuation-in-part of Ser. No. US 1987-105636, filed

<--

on 6 Oct 1987, now abandoned

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Waddell, Frederick E.

ASSISTANT EXAMINER: Fay, Zohreh A.

LEGAL REPRESENTATIVE: Crowley, Steven R.; Weinstock, Steven F.

NUMBER OF CLAIMS: 7
EXEMPLARY CLAIM: 1
LINE COUNT: 3000

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5036051 19910730 <-SUMM Glaucoma is a condition characterized by an increase in intraocular pressure. Increased intraocular pressure can lead to optic nerve damage and defects in the visual field. Blindness can result if the condition is left untreated.

SUMM . . . such as dextran, hydroxyloweralkyl dextran, carboxymethyl dextran, hydroxyloweralkyl cellulose, loweralkyl cellulose, carboxymethyl cellulose, polyvinyl alcohol, dextrin, starch, polyvinyl pyrrolidone and polyalkylene glycols may be used as the carrier for the drug.

L17 ANSWER 105 OF 117 USPATFULL

Substituted imidazolyl-alkyl-piperazine and -diazepine derivatives

ACCESSION NUMBER:

91:32446 USPATFULL

TITLE:

Substituted imidazolyl-alkyl-piperazine and -diazepine

derivatives

INVENTOR(S):

Pascal, Jean C., Cachan, France

Lee, Chi-Ho, Palo Alto, CA, United States

Alps, Brian J., Linlithgow, Scotland

Pinhas, Henri, Paris, France

Whiting, Roger L., Los Altos, CA, United States Syntex Pharmaceuticals Ltd., Berkshire, England

PATENT ASSIGNEE(S):

(non-U.S. corporation)

DATE NUMBER _____

PATENT INFORMATION:

US 5010075 19910423

APPLICATION INFO .:

US 1990-505379 19900406 (7)

RELATED APPLN. INFO.:

Division of Ser. No. US 1989-313656, filed on 21 Feb 1989, now patented, Pat. No. US 4935417 which is a division of Ser. No. US 1987-42181, filed on 24 Apr

1987, now patented, Pat. No. US 4829065

DOCUMENT TYPE:

Utility

PRIMARY EXAMINER: ASSISTANT EXAMINER:

Shah, Mukund J. Dalton, Philip I.

LEGAL REPRESENTATIVE:

Lowin, David A.; Moran, Tom M.

NUMBER OF CLAIMS: EXEMPLARY CLAIM:

13 1,10

LINE COUNT:

1450

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 5010075 19910423 PΤ

. . . a variety of disease states, such as stroke, epilepsy, SUMM hypertension, angina, migraine, arrhythmia, thrombosis, embolism and also for treatment of spinal injuries.

. . . a variety of disease states, such as stroke, epilepsy, SUMM

hypertension, angina, migraine, arrhythmia, thrombosis, embolism and also for treatment of spinal injuries, comprising

administering a therapeutically effective amount of compound of Formula

A to a mammal.

. . . include stroke, migraine, epilepsy, hypertension, angina, SUMM arrhythmia, thrombosis, and embolism. The compounds of this invention are also useful for treating spinal injuries, and are particularly useful for treating cerebrovascular disease states,

for

example, stroke.

For systemic administration via suppository, traditional binders and SUMM carriers include, for example, polyalkaline glycol or triglycerides [e.g., PEG 1000 (96%) and PEG 4000 (4%)]. Such suppositories may be formed from mixtures containing active ingredients in the range of from about 0.5 wt/%. . .

ANSWER 106 OF 117 USPATFULL

Parenteral formulations of 1-diphenylmethyl-4-((2-(4-methylphenyl)-5methyl-1H-imidazol-4-yl)methyl)piperazine

ACCESSION NUMBER:

90:91103 USPATFULL

TITLE:

Parenteral formulations of 1-diphenylmethyl-4-((2-(4-

methylphenyl)-5-methyl-1H-imidazol-4-

yl)methyl)piperazine

INVENTOR(S):

Selkirk, Alastair B., Edinburgh, Scotland Dey, Michael J., West Lothian, Scotland

PATENT ASSIGNEE(S):

Syntex Pharmaceuticals, Ltd., Maidenhead, England

(non-U.S. corporation)

```
NUMBER
                                          DATE
                         _____
PATENT INFORMATION:
APPLICATION INFO.:
                        US 4973591 19901127
                                                                      <--
                        US 1988-260628 19881021 (7)
DOCUMENT TYPE:

PRIMARY EXAMINER:

ASSISTANT EXAMINER:

LEGAL REPRESENTATIVE:

LOWIN, David A.; Moran, Tom M.
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM:
                        463
LINE COUNT:
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       US 4973591 19901127
       diseases treated by direct neuronal protection, such as ischemia
SUMM
       including focal and global ischemia, spinal injuries
       , head trauma, and neurological diseases such as Alzheimer's and
       Huntington's chorea;
       . . . of aqueous miscible cosolvents commonly used to improve the
SUMM
       solubility of parenteral products [for example, various mixtures of
       polyethylene glycol (PEG 300), propylene glycol (PG) and
       ethanol], achieved limited increase in the solubility of the active
       agent. However, all of the. . .
       diseases treated by direct neuronal protection, such as ischemia
SUMM
       including focal and global ischemia, spinal injuries
       , head trauma, and neurological diseases such as Alzheimer's and
       Huntington's chorea;
       diseases treated by direct neuronal protection, such as ischemia
SUMM
       including focal and global ischemia, spinal injuries
       , head trauma, and neurological diseases such as Alzheimer's and
       Huntington's chorea;
L17 ANSWER 107 OF 117 USPATFULL
     Stoma measuring device
ACCESSION NUMBER:
                       90:90358 USPATFULL
TITLE:
                        Stoma measuring device
                        Iversen, Kent, Columbus, OH, United States
INVENTOR(S):
                        Isaac, Ronald, Worthington, OH, United States
                        Abbott Laboratories, Abbott Park, IL, United States
PATENT ASSIGNEE(S):
                         (U.S. corporation)
                             NUMBER DATE
                         ______
PATENT INFORMATION: US 4972845 19901127 APPLICATION INFO.: US 1989-293860 19890105 (7)
                                                                     <--
DOCUMENT TYPE:
                        Utility
PRIMARY EXAMINER:
PRIMARY EXAMINER: Green, Randall L.
ASSISTANT EXAMINER: Reichle, Karin
LEGAL REPRESENTATIVE: Nickey, D. O.; Gorman, E. H.; Phillips, Patrick
                        15
NUMBER OF CLAIMS:
EXEMPLARY CLAIM:
                        4 Drawing Figure(s); 3 Drawing Page(s)
NUMBER OF DRAWINGS:
LINE COUNT: `
                         348
       US 4972845 19901127
ΡI
       . . . are unable to ingest enough solid food to meet their body's
       needs. Examples of these individuals would include stroke or
     neurologically impaired patients, who have lost their
       ability to swallow effectively; critically ill, weak or comatose
       patients, who may be unable to. . .
      . . . surgical procedure utilizing a general anesthetic, the
SUMM
```

preferred method for placement of these ports is through a percutaneous endoscopic gastrostomy (PEG) which involves the non-invasive surgical creation of an artificial opening into the stomach through the abdominal wall using only a local anesthetic. In a PEG procedure, an endoscope is passed down the throat until its terminus contacts the interior of the stomach. A needle is. . .

L17 ANSWER 108 OF 117 USPATFULL

Sustituted imidazolyl-alkyl-piperazine and -diazepine derivatives for treating cerebrovascular disease

ACCESSION NUMBER:

90:48802 USPATFULL

TITLE:

Sustituted imidazolyl-alkyl-piperazine and -diazepine

derivatives for treating cerebrovascular disease

INVENTOR(S):

Pascal, Jean C., Cachan, France

Lee, Chi-Ho, Palo Alto, CA, United States Alps, Brian J., Linlithgow, Scotland

Pinhas, Henri, Paris, France

Whiting, Roger L., Los Altos, CA, United States Syntex Pharmaceuticals Ltd., Hamilton, Bermuda

PATENT ASSIGNEE(S):

(non-U.S. corporation)

NUMBER DATE ______

PATENT INFORMATION:

US 4935417 19900619

APPLICATION INFO.:

US 1989-313656 19890221 (7)

RELATED APPLN. INFO.:

Division of Ser. No. US 1987-42181, filed on 24 Apr

1987, now patented, Pat. No. US 4829065

DOCUMENT TYPE:

Utility

PRIMARY EXAMINER:

Friedman, Stanley J.

LEGAL REPRESENTATIVE: Lowin, David A.; Moran, Tom M.

NUMBER OF CLAIMS: EXEMPLARY CLAIM:

17

LINE COUNT:

1480

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 4935417 19900619 PΙ

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PARN

. . . a variety of disease states, such as stroke, epilepsy, hypertension, angina, migraine, arrhythmia, thrombosis, embolism and

also for treatment of spinal injuries.

. . . a variety of disease states, such as stroke, epilepsy, PARN hypertension, angina, migraine, arrhythmia, thrombosis, embolism and also for treatment of spinal injuries, comprising administering a therapeutically effective amount of compound of Formula A to a mammal.

. . . include stroke, migraine, epilepsy, hypertension, angina, PARN arrhythmia, thrombosis, and embolism. The compounds of this invention are also useful for treating spinal injuries, and are particularly useful for treating cerebrovascular disease states,

for

example, stroke.

For systemic administration via suppository, traditional binders and PARN carriers include, for example, polyalkaline glycol or triglycerides [e.g., PEG 1000 (96%) and PEG 4000 (4%)]. Such suppositories may be formed from mixtures containing active ingredients in the range of from about 0.5 wt/%. . .

L17 ANSWER 109 OF 117 USPATFULL

Glaucoma treatment

90:40541 USPATFULL ACCESSION NUMBER: Glaucoma treatment TITLE:

Stein, Herman H., Highland Park, IL, United States INVENTOR(S): Pattner, Jacob J., Libertyville, IL, United States

Crowley, Steven R., Vernon Hills, IL, United States Abbott Laboratories, Abbott Park, IL, United States PATENT ASSIGNEE(S):

(U.S. corporation)

DATE NUMBER

US 4927807 19900522 US 1988-240567 19880908 (7) PATENT INFORMATION:

APPLICATION INFO.:

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1987-105636, filed

on 6 Oct 1987, now abandoned

DOCUMENT TYPE: Utility

Robinson, Douglas W. PRIMARY EXAMINER:

Fay, Zohreh A. ASSISTANT EXAMINER:

LEGAL REPRESENTATIVE: Crowley, Steven R.; Weinstock, Steven F.

NUMBER OF CLAIMS: EXEMPLARY CLAIM: LINE COUNT: 2997

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 4927807 19900522 SUMM

Glaucoma is a condition characterized by an increase in intraocular pressure. Increased intraocular pressure can lead to optic nerve damage and defects in the visual field. Blindness can result if the condition is left untreated.

SUMM . . . such as dextran, hydroxyloweralkyl dextran, carboxymethyl dextran, hydroxyloweralkyl cellulose, loweralkyl cellulose, carboxymethyl cellulose, polyvinyl alcohol, dextrin, starch, polyvinyl pyrrolidone and polyalkylene glycols may be used as the carrier for the drug.

L17 ANSWER 110 OF 117 USPATFULL

Treatment of mammals suffering from damage to the central nervous

ACCESSION NUMBER: 89:65118 USPATFULL

TITLE: Treatment of mammals suffering from damage to the

central nervous system

INVENTOR(S): Naftchi, Nosrat E., 389 Forest Ave., Teaneck, NJ,

United States 07666

NUMBER DATE

PATENT INFORMATION: US 4855325 19890808 US 1988-150767 19880201 (7) APPLICATION INFO.:

20050503 DISCLAIMER DATE:

Division of Ser. No. US 1985-691830, filed on 16 Jan RELATED APPLN. INFO.:

1985, now patented, Pat. No. US 4742054 which is a continuation of Ser. No. US 1982-443915, filed on 23

<--

Nov 1982, now abandoned

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Rollins, John W. LEGAL REPRESENTATIVE: Magidoff, Barry G.

NUMBER OF CLAIMS: EXEMPLARY CLAIM: 1 LINE COUNT: 546

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 4855325 19890808

. . . been discovered that by the use of a neural receptor agonist, SUMM e.g., clonidine, many of the undesirable aftereffects of traumatic

spinal injury can be alleviated or completely

eliminated, and, if treatment is commenced sufficiently early, at least

some restoration of normal neural. . .

. . . include water, gelatine, lactose, starches, stearic acid, SUMM

magnesium stearate, sicaryl alcohol, talc, vegetable oils, benzyl alcohols, gums, waxes, propylene glycol, polyalkylene glycols or any other know carrier for medicaments.

L17 ANSWER 111 OF 117 USPATFULL

Substituted imidazolyl-alkyl-piperazine and -diazepine derivatives

ACCESSION NUMBER: 89:36735 USPATFULL

TITLE:

Substituted imidazolyl-alkyl-piperazine and -diazepine

derivatives

INVENTOR (S):

Pascal, Jean C., Cachan, France

Lee, Chi-Ho, Palo Alto, CA, United States Alps, Brian J., Linlithgow, Scotland

Pinhas, Henri, Paris, France

Whiting, Roger L., Los Altos, CA, United States Syntex Pharmaceuticals, Ltd., Maidenhead, England

(non-U.S. corporation)

NUMBER _____

PATENT INFORMATION:

PATENT ASSIGNEE(S):

US 4829065 19890509

APPLICATION INFO.:

US 1987-42181 19870424 (7)

DOCUMENT TYPE:

Utility

PRIMARY EXAMINER: Friedman, Stanley J.

LEGAL REPRESENTATIVE: Lowin, David A.; Moran, Tom M.

NUMBER OF CLAIMS: 14 EXEMPLARY CLAIM:

LINE COUNT:

1470

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

US 4829065 19890509 PΙ

<--

SUMM . . . a variety of disease states, such as stroke, epilepsy, hypertension, angina, migraine, arrhythmia, thrombosis, embolism and also for treatment of spinal injuries.

. . . a variety of disease states, such as stroke, epilepsy, SUMM hypertension, angina, migraine, arrhythmia, thrombosis, embolism and also for treatment of spinal injuries, comprising administering a therapeutically effective amount of compound of Formula

A to a mammal.

. . . include stroke, migraine, epilepsy, hypertension, angina, SUMM arrhythmia, thrombosis, and embolism. The compounds of this invention are also useful for treating spinal injuries, and are particularly useful for treating cerebrovascular disease states,

for

example, stroke.

For systemic administration via suppository, traditional binders and SUMM carriers include, for example, polyalkaline glycol or triglycerides [e.g., PEG 1000 (96%) and PEG 4000 (4%)]. Such suppositories may be formed from mixtures containing active ingredients in the range of from about 0.5 wt/%. . .

L17 ANSWER 112 OF 117 USPATFULL

TΙ Treatment of mammals suffering from damage to the central nervous system

ACCESSION NUMBER:

88:27758 USPATFULL

TITLE:

Treatment of mammals suffering from damage to the

central nervous system

INVENTOR(S):

Naftchi, Nosrat E., 389 Forest Ave., Teaneck, NJ,

United States 07666

NUMBER DATE _____

PATENT INFORMATION:

US 4742054 19880503

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APPLICATION INFO.: US 1985-691830 19850116 (6) Continuation of Ser. No. US 1982-443915, filed on 23 RELATED APPLN. INFO.: Nov 1982, now abandoned DOCUMENT TYPE: Utility PRIMARY EXAMINER: Brown, J. R. ASSISTANT EXAMINER: Rollins, Jr., John W. Magidoff, Barry G. LEGAL REPRESENTATIVE: NUMBER OF CLAIMS: 23 EXEMPLARY CLAIM: LINE COUNT: 581 CAS INDEXING IS AVAILABLE FOR THIS PATENT. US 4742054 19880503 SUMM . . . been discovered that by the use of a neural receptor agonist, e.g., clonidine, many of the undesirable aftereffects of traumatic spinal injury can be alleviated or completely eliminated, and, if treatment is commenced sufficiently early, at least some restoration of normal neural. . . SUMM . . . include water, gelatine, lactose, starches, stearic acid, magnesium stearate, sicaryl alcohol, talc, vegetable oils, benzyl alcohols, gums, waxes, propylene glycol, polyalkylene glycols or any other known carrier for medicaments. L17 ANSWER 113 OF 117 USPATFULL Wheeled seat carrying apparatus and stroller for the handicapped ACCESSION NUMBER: 88:14268 USPATFULL TITLE: Wheeled seat carrying apparatus and stroller for the handicapped INVENTOR(S): Bergeron, Timothy J., R.D. 1, Box 40, Dolgeville, NY, United States 13329 DATE NUMBER _____ PATENT INFORMATION: US 4729572 19880308 <--US 1987-32222 19870330 (7) APPLICATION INFO.: DOCUMENT TYPE: Utility PRIMARY EXAMINER: Love, John J. ASSISTANT EXAMINER: Mar, Michael LEGAL REPRESENTATIVE: Heslin & Rothenberg 29 NUMBER OF CLAIMS: EXEMPLARY CLAIM: 1 NUMBER OF DRAWINGS: 8 Drawing Figure(s); 7 Drawing Page(s) LINE COUNT: 729 US 4729572 19880308 SUMM . . strollers for handicapped individuals and more particularly, to an adjustable wheeled apparatus designed to carry a seat support for a neurologically impaired child, adolescent or adult. Neurologically impaired individuals suffer from injury, disease or disorder of the brain or nervous system. Two leading causes of neurological impairment, particularly in children and adolescents, are cerebral palsy and muscular dystrophy. Although the severity of such disorders will vary, in. . . such as partial or total loss of muscular control and motion, and partial loss of speech, hearing and reasoning abilities. Neurological impairment and its effects are discussed in some detail in a copending application entitled "Seat Support and Restraint System for SUMM . . reside within the receiving channels defined by the parallel plate pairs. The second frame includes an externally protruding spring

biased peg near one free end which is positioned to

selectively engage the holes aligned in the arc-shaped configuration in

the one. . .

SUMM The second frame is locked in position relative to the first frame when the spring biased **peg** engages one of the holes arranged in the arc-shaped configuration in the one parallel plate and the angle at which. . . axis of the second frame intersects the axis of the first frame is selectively varied by moving the spring biased **peg** such that the **peg** engages a different one of the holes. The distance between the wheels secured to the first frame and the wheels.

L17 ANSWER 114 OF 117 USPATFULL

TI EDUCATIONAL APPARATUS

ACCESSION NUMBER: 73:17011 USPATFULL EDUCATIONAL APPARATUS

INVENTOR(S): Magram, David, 2304 Sherwood St., Pittsburgh, PA,

United States 15217

NUMBER DATE

PATENT INFORMATION: US 3728800 19730424 <--

APPLICATION INFO.: US 1971-180659 19710915 (5)

DOCUMENT TYPE: Utility PRIMARY EXAMINER: Grieb, Wm. H.

LEGAL REPRESENTATIVE: Stein; Arland T.; Wettach; Thomas C.; Yeager; Robert

D.

NUMBER OF CLAIMS: 5

NUMBER OF DRAWINGS: 17 Drawing Figure(s); 3 Drawing Page(s)

LINE COUNT: 239

PI US 3728800 19730424 <--

SUMM . . . of teaching language, which is analytical, has not been

particularly successful with young children, particularly deaf children or those with neurological impairments, foreign

students, etc. These children often require special assistance in learning the language patterns and the traditional techniques are

rarely. . .

SUMM . . . may be advantageously used with the older group of students. I provide a linkage system which includes a combination of **pegs** arranged in a geometrical pattern that fit holes in a complementary pattern in a block to be aligned and fitted. . .

L17 ANSWER 115 OF 117 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
TI Sterile aq. lazaroid compsn. for treatment of head and spinal injuries
etc. - is admin. parenterally and also comprises citrate, co-solvent and
water.

ACCESSION NUMBER: 1996-160131 [16] WPIDS

DOC. NO. CPI: C1996-050516

TITLE: Sterile aq. lazaroid compsn. for treatment of head and

spinal injuries etc. - is admin. parenterally and also

comprises citrate, co-solvent and water.

DERWENT CLASS: B01

INVENTOR(S): BAKER, D S; MACHKOVECH, S M; SU, C; SU, C C PATENT ASSIGNEE(S): (UPJO) UPJOHN CO; (PHAA) PHARMACIA & UPJOHN CO

COUNTRY COUNT: 66

PATENT INFORMATION:

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WO 9606618 A1 19960307 (199616) * EN 18 <--

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PRAI US 1995-382256
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AΒ
     (1) 0.9-90 mg/ml lazaroid or its salts; (2) 0.002-2.0 M citrate; (3) up
to
     80% cosolvent selected from propylene glycol, polyethylene
     qlycol, glycerol, ethanol, DMSO, DMAC, DMI and M-PYROL; and (3)
     water at pH 2.4-3.5.
          Lazaroids are useful in treating and/or preventing spinal
     injury, head injury, subarachnoid haemorrhage and subsequent
     ischaemic stroke, asthma and redn. of mucous formation/secretion in the
     lung, muscular dystrophy, adriamycin. . . colitis and Crohn's disease.
     Lazaroids are also useful for prophylactic treatment before surgical
     procedures where they reduce oedema, for preventing neurologic
     injury during surgical and neurological procedures, for treatment
     of myocardial infarction, for treatment after resuscitation to improve
     outcome, for treatment of.
    ANSWER 116 OF 117 WPIDS COPYRIGHT 2001
                                               DERWENT INFORMATION LTD
    ATP-sensitive potassium channel blocker - useful for treatment of
neuronal
     insult in brain due to lack of oxygen to prevent Parkinsonian
     degeneration.
                      1992-026514 [04]
                                         WPIDS
ACCESSION NUMBER:
DOC. NO. CPI:
                      C1992-011399
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ATP-sensitive potassium channel blocker - useful for treatment of neuronal insult in brain due to lack of

oxygen to prevent Parkinsonian degeneration.

TITLE:

DERWENT CLASS:

B05

INVENTOR(S): MURPHY, K P S; GREENFIELD, S A; MURPHY, K P S J
PATENT ASSIGNEE(S): (MURP-I) MURPHY K P S J; (SQUI) SQUIBB & SONS INC E R
COUNTRY COUNT: 7

PATENT INFORMATION:

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FILING DETAILS:

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PRAI US 1990-556502 19900720; US 1992-826546 19920127; US 1993-31506
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                   UPAB: 19931119
     Pharmaceutical (I) which blocks an ATP-sensitive K channel in the brain
is
     used in compsns. for treating neuronal damage in the
     brain caused by a lack of O2.
           (I) can be admin., e.g. by infusion, to the substantia nigra, . . .
     Typical soln. for infusion e.g. by lumbar puncture, contains 250 g
     tolbutamide and 25 g NaCl dissolved in 1.5 l polyethylene
     glycol 400 plus enough water for injection to make 51.
          USE/ADVANTAGE - (I) are used to treat the early stages of. . .
L17 ANSWER 117 OF 117 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
     Transporting stretcher for injured person - has non-metallic middle part
     permeable to X-rays.
ACCESSION NUMBER: 1989-370524 [50] WPIDS
DOC. NO. NON-CPI: N1989-282035
TITLE: Transporting stretcher for injured person - has
                      non-metallic middle part permeable to X-rays.
DERWENT CLASS: P31 P33
INVENTOR(S): FICKLER, H
PATENT ASSIGNEE(S): (FICK-I) FICKLER H
COUNTRY COUNT:
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FILING DETAILS:

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    Publ. HU 54484, Based on WO 8911263
PRAI CH 1988-2009
                  19880527
ABEO.
    the support form a recess within which the retaining block is positioned.
    The support has a bore. A removably spring-loaded peg is
    normally urged within the bore into a locked position between the support
    and the retaining block.
         USE - For people with spinal injuries.
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Executing the logoff script...
=> LOG Y
COST IN U.S. DOLLARS
                                               SINCE FILE
                                                              TOTAL
                                                   ENTRY
                                                            SESSION
FULL ESTIMATED COST
                                                  4719.59
                                                            4791.21
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)
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                                                              TOTAL
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-0.59

-0.59

CA SUBSCRIBER PRICE

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4	BRS	17560	blocker or antagonist	USPAT	2000/09/29 12:49	•		0
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7	BRS	30411	nery? or neur?	USPAT; EPO; JPO; Derwent; IBM TDB	2000/12/21 15:24			0
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12	BRS	99	(potassium adj channel) adj (blocker or antagonist)	USPAT	2000/12/22 11:38			0
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	BRS	33719	((polybropylene adj glycol) or (polybutylene adj glycol) or (polypentylene adj glycol) or (polyhexylene adj glycol) or (polyheptylene adj glycol) or (polyoctylene adj glycol)) or (polyalkylene adj glycol)	USPAT; EPO; JPO; Derwent; IBM TDB	2001/02/01 13:18			0
	BRS	4975	(spin\$ or axon\$ or neuro\$) with (injury or injuries or damagae or impair\$)	USPAT	2001/02/01 13:14		Truncation Overflow. Return string from Server is: 5.0.0.SPI	1

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32	BRS	_	(((polypropylene adj glycol) or (polybutylene adj glycol) or (polypentylene adj glycol) or (polyhexylene adj glycol) or (polyheptylene adj glycol) or (polyoctylene USI adj glycol)) or (polyalkylene adj glycol)) same ((spin\$ or axon\$ or neuro\$) with (injury or injuries or damagae or impair\$))	USPAT	2001/02/01 13:08			0
33	BRS	155	(((polybropylene adj glycol) or (polybutylene adj glycol) or (polypentylene adj glycol) or (polyhexylene adj glycol) or (polyheptylene adj glycol) or (polyoctylene USI adj glycol)) or (polyalkylene adj glycol)) and ((spin\$ or axon\$ or neuro\$) with (injury or injuries or damagae or impair\$))	USPAT	2001/02/01 13:13			0
34	BRS	155	((((polypropylene adj glycol) or (polybutylene adj glycol) or (polypentylene adj glycol) or (polyhexylene adj glycol) or (polyheptylene adj glycol) or (polyoctylene adj glycol)) or (polyalkylene adj glycol)) and ((spin\$ or axon\$ or neuro\$) with (injury or injuries or damagae or impair\$))) and (py<1998)	USPAT	2001/02/01 13:14	•	_	0
35	BRS	6294	(spinal or spine or axon\$ or neuron\$ or USI nerve) with (injury or injuries or damagae Der or impair\$)	USPAT; EPO: JPO; Derwent; IBM TDB	2001/02/01 13:17		; ;	0
36	BRS	155	(((polybutylene adj glycol) or (polypentylene adj glycol) or (polybutylene adj glycol) or (polypentylene adj glycol) or (polyhexylene adj glycol) or (polyhetylene adj glycol) or (polyactylene USPAT; EPO; JPO, adj glycol)) or (polyalkylene adj glycol)) Derwent; IBM TDB and ((spinal or spinc or axon\$ or neuron\$ or nerve) with (injury or injuries or damagae or impair\$))	USPAT; EPO; JPO, Derwent; IBM TDB	2001/02/01 13:26	•		0
37	BRS		(((polypropylene adj glycol) or (polybutylene adj glycol) or (polypentylene adj glycol) or (polypentylene adj glycol) or (polyheptylene adj glycol) or (polyheptylene adj glycol) or (polyalkylene adj glycol)) or (polyalkylene adj glycol)) Der same ((spinal or spine or axon\$ or neuron\$ or nerve) with (injury or injuries or damagae or impair\$))	USPAT; EPO, IPO; Derwent; IBM TDB	2001/02/01 13:26	1		

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